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THE ELECTROCARDIOGRAPHIC DIAGNOSIS OF ACUTE CARDIAC INFARCTION WITH SPECIAL REFERENCE TO THE VALUE OF PRECORDIAL LEADS*

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THE decision as to whether a patient is suffering from acute cardiac infarction is a momentous and often a difficult one. Therefore, any refinement of technique which promises more ready recognition of the condition is worthy of careful study and evaluation. The recent employment of chest leads in the electrocardiographic diagnosis of cardiac infarction has yielded encouraging results which have prompted this investigation.

The history of the clinical study of coronary thrombosis is a brief one. In 1912, J. B. Herrick[1] gave the first description in English of the clinical features of sudden obstruction of the coronary arteries. The same gifted observer called attention to abnormalities in the electrocardiogram following coronary thrombosis[2] and suggested to Fred M. Smith the study of experimental coronary occlusion in dogs by which the electrocardiographic changes seen in human cases were reproduced[3]. Smith showed that these changes have marked variations from day to day, and even from hour to hour, and may eventually disappear.

In 1920, Pardee[4] called attention to a particular form of inversion of T-waves, i.e., late, sharp inversion with upward convexity of the RS-T interval which was frequently associated with occlusion of coronary arteries as shown at postmortem.

However, interest in the clinical diagnosis grew rather slowly. For example, in 1924 only three cases which had received the diagnosis of coronary thrombosis during life had come to autopsy at the Massachusetts General Hospital[5].

In 1928, Parkinson and Bedford[6] called attention to a definite sequence of changes in the RS-T segment and the T-waves of the electrocardiogram following cardiac infarction. "Shortly after the onset of symptoms a transient deviation of the R-T segment from the iso-electric plane occurs. This is followed by a deep inversion of the T-waves in either lead I or lead III, but not in both, and often by a lesser degree of T inversion in lead II. Curves obtained after a few weeks conform to one of two main types, according to the incidence of T inversion in lead I or in lead III. Subsequent T wave changes in the direction of normal are recorded, and even complete return to normal occurs." These authors speculated as to the possibility of correlating the site of the infarct with a specific type of electrocardiographic abnormality but drew no conclusions from their limited autopsy material.

In the following year Barnes and Whitten[7] found that the electrocardiographic changes of the T^1 type described by Parkinson and Bedford were usually associated with infarction of the anterior wall of the left ventricle, and the T^3 type with infarction of the posterior wall of the left ventricle. They observed, as others had before, that infarction of the right ventricle was rare.

More recently the effect of cardiac infarction on the initial deflections of the ventricular complex have received attention. Pardee has pointed out that the incidence of heart disease, particularly the anginal syndrome, is high in individuals exhibiting a large Q-wave in lead III in the presence of a normal electrical axis or left axis deviation[8]. He presented evidence which suggested that the large Q-III is due to disease of the left ventricle. A refinement of his criteria has added to the diagnostic value of Q-wave changes by excluding many normal individuals[9], and their importance in relation to cardiac infarction has been amply confirmed by others, notably Wilson and his coworkers[10]. Wilson's observations indicate that deviations of the R-T interval are due to currents of injury set up at the borders of injured cardiac muscle whereas the changes in the initial ventricular complex (Q-R-S) are explained by the death of areas of formerly active tissue. The former is, therefore, a transient and the latter, a permanent change.

The most recent important contribution to the electrocardiographic diagnosis of cardiac infarction was the introduction of the chest lead by Wolferth and Wood in 1932. Although chest electrodes had been employed by others previously, they were the first to demonstrate clearly that with this technique many cases of

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fresh infarction show electrocardiographic changes which do not appear in the standard limb leads. The changes in the chest lead have also been considered to be of value in localizing the site of the infarct.

In taking the chest lead, Wolferth and Wood placed the right arm electrode over the precordium* and the left arm electrode on the back of the chest just below and mesial to the left scapular angle. This lead they designated as lead IV. In subsequent studies they also made observations on an electrocardiographic lead taken from the precordium to the left leg which they designated as lead V, and on a lead taken from the posterior electrode to the left leg which they designated as lead VI. Comparison of the results derived from the three chest leads showed that leads IV and V were very similar in appearance and lead VI yielded little, if any, additional information of importance. Since their original publication, the clinical use of chest leads in the diagnosis of cardiac infarction has been made in a number of clinics. The practical value of the method has been confirmed by others and its theoretical validity soundly established. The subject has been somewhat confused by the fact that many subsequent investigators have used slightly or widely different parts of the body for placing the electrodes so that their results are not comparable one with another. While it is quite possible that one or more chest leads may be found which will yield even more information than those employed by Wolferth and Wood, their series, together with others using the same technique, represent the largest and best controlled from the point of view of autopsy protocols as well as for normal individuals. It appears, from their own observations, that in most cases all of the information desired can be derived from one of the two precordial leads. As pointed out by Wilson[12], it is the electrode nearest the heart which is the predominating factor in inscribing the electrocardiogram in chest leads. The position of the distal electrode is therefore of relatively minor importance. The records of Wolferth and Wood's lead IV (anteroposterior) are almost identical with their lead V (precordium to left leg) and, for practical purposes, the two may be used almost interchangeably. Since lead V is a little easier to take technically, it is perhaps, of the two, the method of choice. An excellent critical review of the subject of chest leads appears in a recent article by Roth[13].

The amount of information to be derived from precordial leads not yet being fully established, it seemed worthwhile to report on our experience with this procedure at the Boston City Hospital with 250 individuals, in all of whom there was a question of recent coronary thrombosis. The precordial lead employed in almost all of these was "lead V", only a few at the beginning being "lead IV". We have evaluated these records by the criteria set forth by Wood and Wolferth and more accurately defined as regards normals by Master[14]. The ventricular complex in the precordial lead as thus defined consists of an initial downwardly directed wave, the Q-wave, followed by an upward R-wave, an isoelectric interval and, finally, a negative T-wave. The Q-wave is normally at least 1.5 mm. in depth and the R-wave at least 2.5 mm. in height. The R-T interval is either slightly below the isoelectric level or just isoelectric. In normal persons it is never above the isoelectric level or more than 2 mm. below it.

The changes observed in the precordial lead following acute cardiac infarction may be divided into two main types corresponding with the T^1 type and the T^3 type described by Parkinson and Bedford.

In the first type, the Q-wave of the precordial lead either disappears or is markedly diminished in size, and the R-T interval is at first markedly depressed. The depression of the R-T interval persists for from five to nine days and gradually disappears. A high peaked upright T-wave develops and tends to remain (see figure 1).

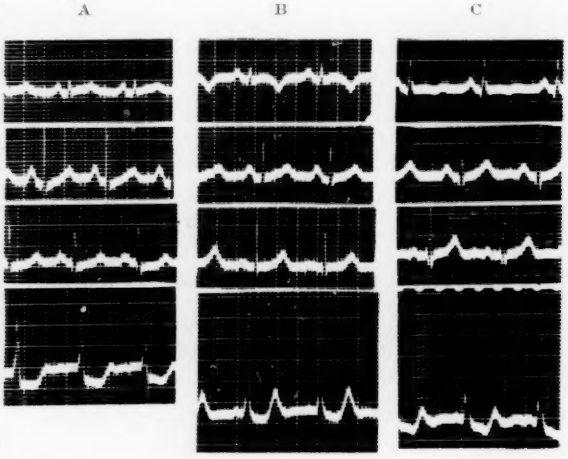

FIGURE 1. Electrocardiogram of the T^1 type. Leads I, II, III and V. A. 3 days, B. 19 days and C. 9 months after coronary thrombosis.

In the second type, the precordial lead is often normal although it may show elevation of the R-T interval or upright or flat T-waves. Changes when they do occur are not usually permanent (see figure 2).

Before proceeding to the analysis of our cases, it may be pointed out that the possibility of factors other than myocardial infarction operating to affect the electrocardiogram have been ruled out as far as possible. By confining this study to the diagnosis of acute infarction, we have avoided the difficult problem of distin-

*The exact position of the precordial electrode is important. For the sake of technical simplicity and uniformity in this series a point 4 cm. to the left of the mid-sternum in the fourth intercostal space was used.

guishing between electrocardiographic changes due to old infarction and those due to myocardial damage from other causes. This has limited the electrocardiographic criteria to (1) deviations of the R-T segment or (2) marked variations in the ventricular complex from day to day. One condition, other than cardiac infarction which may be associated with deviation of the R-T interval is inflammation of the pericardium, such as tuberculous pericarditis. In this instance, however, the characteristic feature is an elevation of the R-T interval in all

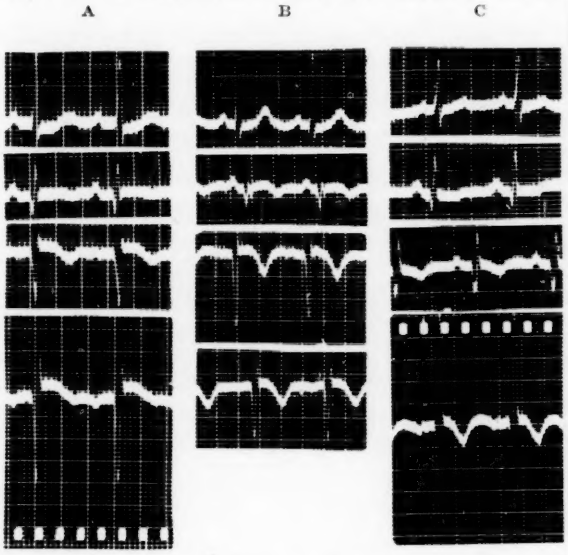

FIGURE 2. Electrocardiogram of the T^3 type. Leads I, II, III and V. A. 6 days, B. 23 days and C. 10 months after coronary thrombosis.

leads rather than in one or two leads only. Other conditions which may produce comparable changes in the electrocardiogram are diphtheritic and other forms of acute myocarditis, massive pulmonary embolism[15] and, perhaps, digitalis intoxication[16].

A comparison of the electrocardiographic interpretation with the final clinical diagnosis is of some interest although, of course, it does not approach in significance a comparison with necropsy findings.

Of the 250 cases studied, fifty-one were interpreted as acute cardiac infarction from the electrocardiogram (see figure 3). Of these, forty-four were so diagnosed clinically, three were classed as suspicious and four as negative. Nine cases had a questionable electrocardiographic interpretation of acute infarction. Of these, four had a positive clinical diagnosis, three a questionable diagnosis and two a negative diagnosis. Of the 190 cases who had no electrocardiographic signs of acute infarction, the clinical diagnosis was in agreement in 163 cases but twenty-four had a positive clinical diagnosis and three were classed as questionable. The discrepancy between the clinical and the electrocardiographic diagnoses is explainable in part by the fact that some of the cases in which a positive clinical diagnosis was made did not have electrocardiograms until after the acute stage of infarction was over and the characteristic electrical changes might be expected to have disappeared.

In thirty-five of the fifty-one cases exhibiting the electrocardiographic signs of cardiac infarction the diagnostic abnormalities were present simultaneously in both the precordial and limb leads. In these instances the chest lead was of value only in confirming the evidence derived from the standard leads. In five cases the changes persisted in the limb leads after the chest lead had returned to normal. In two cases the abnormality appeared only in the standard leads. In one case, in which the clinical picture was unmistakable, the electrocardiographic diagnosis was made only by striking variations in the record from day to day, although no single record was in itself diagnostic.

In eight cases the diagnostic abnormality was present at some time only in the chest lead. All of these cases had a positive clinical diagnosis of coronary thrombosis. In three, the changes appeared only in the chest lead throughout; in two, it preceded the development of changes in the standard leads; in two, it followed them and in one, it both preceded and followed them. This group of eight cases comprising approximately 16 per cent of our total positive diagnoses illustrates the importance of the precordial leads. In three cases out of fifty-one the diagnosis could never have been made without the chest lead.

Complete necropsy records were available on thirty-three cases on which precordial leads had been taken. These records were of routine hospital necropsies performed by a number of different pathologists. The exact extent or localization of the infarct beyond the statement that it was situated in the anterior, apical or posterior portion of the ventricle was not always clearly recorded. This must be borne in mind in evaluating the correlation between the electrocardiographic and the anatomical localization of the infarcted areas. Thirteen cases showed acute cardiac infarction at postmortem. The electrocardiographic diagnosis was made in eleven of these (see figure 3). In each of the two cases in which the electrocardiographic diagnosis was not made, a single record was obtained shortly before death. In one it appeared that the infarction, although recent, was of too long standing to show the characteristic changes in a single record, the history indicating clearly that the thrombosis had occurred nineteen days before the record was taken. It showed an absence of the Q-wave in the precordial lead, a flat T^1, very low T^2 and inverted T^3. In the other, the record was obtained a few hours after the onset and showed intraventricular block. In both there was infarction of the anterior wall of the left ventricle, and both showed

marked coronary sclerosis with extensive myocardial scarring.

Of the thirteen cases with acute infarction, twelve were in the anterior or apical portion of the left ventricle and one in the posterior wall. The anterior infarcts were associated with the following electrocardiographic findings:—five with changes of the T^1 type, three with changes of the T^3 type, two with changes of a nonspecific type, and two with signs of old coronary disease only. The one case with posterior infarction showed electrocardiographic changes of the T^3 type.

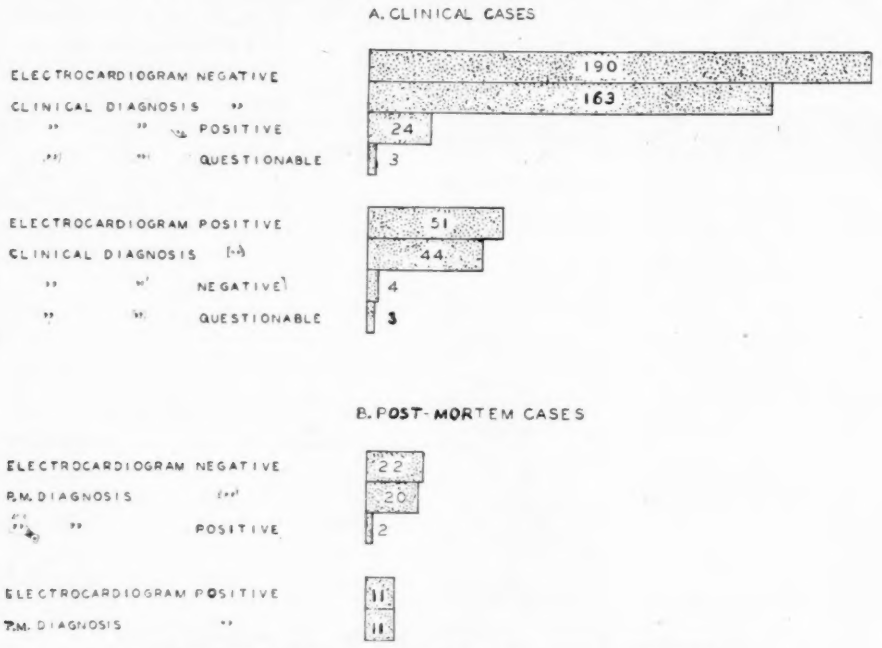

FIGURE 3. Correlation between the electrocardiographic findings and the clinical and postmortem findings in acute cardiac infarction (9 cases in which the electrocardiographic diagnosis was questionable are not shown in this chart).

In this series the correlation was better between the electrocardiographic type and the artery occluded than with the actual site of the infarct. All of five instances of the T^1 type were associated with occlusion of the anterior descending branch of the left coronary artery and three out of four of the T^3 type had occlusion of the circumflex branch.

In the remaining twenty autopsied cases in which there was no infarction, a questionable electrocardiographic diagnosis was made in four. One of these had left bundle branch block with a markedly depressed R-T interval in the chest lead (7 mm.). Another had intraventricular block with an elevated R-T level in the chest lead (3.5 mm.). The third case showed an auricular tachycardia with two to one heart block and a ventricular rate of 115. The take-off of T^5 was 4 mm. below the baseline. The fourth case showed inversion of T^1 with low origin and upward convexity of S-T on two records taken two days apart, and three days later showed an upright T^1—still with low origin. This patient died two days after the last record was taken and showed a dissecting aneurysm of descending thoracic aorta without involvement of the coronary arteries or pericardium. It seems obvious that there will always be instances of borderline or doubtful changes in the electrocardiogram which cannot be interpreted with certainty. The cases which are most difficult to evaluate are those in which there is electrocardiographic evidence of old myocardial disease and, particularly, bundle branch block.

SUMMARY AND CONCLUSIONS

In a series of electrocardiograms including precordial leads on 250 patients, a diagnosis of fresh cardiac infarction was made fifty-one times. In eight of these the diagnostic feature was present at some time only in the chest lead, and in three it was never present in the standard leads.

A comparison between the electrocardiographic and the autopsy findings was made in thirty-three cases. Out of thirteen cases with acute infarction the electrocardiographic diagnosis was positive in eleven. Localization of the infarct was not reliable by the method used.

Of the twenty cases who did not show infarction, four had a questionable electrocardiographic diagnosis, and in the remainder it was negative.

The findings here recorded support the following conclusions:

(1) During the first two weeks after the de-

veloping of cardiac infarction the electrocardiogram is a highly accurate method of determining the diagnosis.

(2) The precordial lead offers additional information of definite value in the diagnosis of fresh cardiac infarction.

REFERENCES

1. Herrick, J B.: Clinical features of sudden obstruction of the coronary arteries. J. A. M. A. **59:** 2015 (Dec. 7) 1912.
2. Ibid.: Thrombosis of the coronary arteries. J. A. M. A. **72:** 387 (Feb. 8) 1919.
3. Smith, F. M.: The ligation of coronary arteries with electrocardiographic study. Arch. Int. Med. **22:** 8 (July) 1918.
4. Pardee, H. E. B.: An electrocardiographic sign of coronary artery obstruction. Arch. Int. Med. **26:** 244 (Aug.) 1920.
5. Faulkner, J. M.; Marble, H. C., and White, P. D.: The differential diagnosis of coronary occlusion and of cholelithiasis. J. A. M. A. **83:** 2080 (Dec. 27) 1924.
6. Parkinson, J., and Bedford, D. E.: Successive changes in electrocardiogram after cardiac infarction (coronary thrombosis). Heart **14:** 195 (Aug.) 1928.
7. Barnes, A. R., and Whitten, M. B.: Study of the R-T interval in myocardial infarction. Am. Heart J. **5:** 142 (Dec.) 1929.
8. Pardee, H. E. B.: The significance of an electrocardiogram with a large Q in lead 3. Arch. Int. Med. **46:** 470 (Sept.) 1930.
9. Durant, T. M.: The initial ventricular deflection of the electrocardiogram in coronary disease. Am. J. M. Sc. **188:** 225 (Aug.) 1934.
10. Wilson, F. N.; MacLeod, A. G.; Barker, P. S.; Johnston, F. D., and Klostermeyer, L. L.: Electrocardiogram in myocardial infarction with particular reference to initial deflections of ventricular complex. Heart **16:** 155 (June) 1933.
11. Wolferth, C. C., and Wood, F. C.: The electrocardiographic diagnosis of coronary occlusion by the use of chest leads. Am. J. M. Sc. **183:** 30 (Jan.) 1932.
12. Wilson, F. N.; Johnston, F. D., and Hill, I. G. W.: Interpretation of galvanometric curves obtained when one electrode is distant from heart and other near or in contact with ventricular surface; observations on cold-blooded heart. Am. Heart J. **10:** 163 (Dec.) 1934.
13. Roth, I. R.: On the use of chest leads in clinical electrocardiography. Am. Heart J. **10:** 798 (Aug.) 1935.
14. Master, A. M.: The precordial lead in 104 normal adults. Am. Heart J. **9:** 511 (April) 1934.
15. McGinn, S., and White, P. D.: Acute cor pulmonale resulting from pulmonary embolism; its clinical recognition. J. A. M. A. **104:** 1473 (Apr. 27) 1935.
16. Strauss, H., and Katz, L. N.: Effect of digitalis on appearance of lead IV. Am. Heart J. **10:** 546 (April) 1935.

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SECONDARY CARCINOMATA OF THE LARGE BOWEL*

BY EDWARD L. YOUNG, JR., M.D.†

BY secondary carcinoma of the large bowel I have reference to those cases where, following an operation for the disease, a second growth occurs in the colon at some distance from the primary tumor.

The etiology of carcinoma of the colon, just as in all malignant tumors, remains obscure. Nevertheless, we do know of certain contributing or predisposing factors, and in particular we recognize the fact that certain types of benign growths may show a malignant degeneration. This is a definitely recognized fact in relation to adenomatous polypi of the large intestine. We have no proof that they all become malignant but certain it is if a patient lives long enough, the vast majority will make trouble of this nature. Neither do we know with certainty that all carcinomata of the large bowel originate in polyps but there is a good deal of evidence to make us think that at least a large proportion of them do. It is, of course, well recognized that these papillomata are very apt to be multiple. In fact the presence of one should put the burden of proof on the individual who tries to prove it is the only one. Although these facts are beyond dispute, I think they have received too little attention in relation to the recurrence of cancer of the colon. When a patient comes back with a recurrence of this disease, it is too often assumed that it is a recurrent manifestation of the first growth when in fact it may be and I think often is entirely separate and just as amenable to treatment if recognized in time as the first tumor. I believe that is the important fact relating to this subject and the justification for this paper. The following cases are illustrative of the problem at hand and I am putting them down in the order in which it seems to me they emphasize the story.

CASE 1. M. G. H., E. S. 281130, a male, aged thirty-eight. He came to the hospital in January, 1927 with the story of one year of intermittent pain in the right lower quadrant. His bowels had been normal until two weeks before entry when they were loose but there was no blood. X-ray showed a tumor of the cecum. At operation a right colectomy was done and the pathological diagnosis was adenocarcinoma of the colloid type with intestinal polyps, inasmuch as the mucous membrane above the growth showed numerous small adenomas from the size of a buckshot to a pea. Those examined did not show any malignancy. He has been followed in the tumor clinic. Shortly after operation he reported an occasional show of blood on the stool but he laid it to hemorrhoids which he undoubtedly had. X-rays were negative through December, 1929, nearly three years, and no rectal examination was done until just after the last x-ray. That examination showed rectal polyps. He was referred to the House where he was seen by Dr. D. F. Jones, in consultation on January 5, 1930, who stated that inasmuch as the patient was known to have had polypi in the ascending colon and rectosigmoid region, he undoubtedly had them between and that he would eventually die of carcinoma if he did not have a total colectomy, but that the operation was of sufficient severity so that if the patient could be made to understand the problem, he should be the one to decide. As a result the visible polyps in the rectum were fulgurated and eight months later a large papilloma of the rectum was excised with the diathermy current. The pathological report on this says, "A growth 4 x 3 x 1½ cm. microscopically showing atypical changes of the epithelium, very suggestive of malignant degeneration

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but no invasive growth and examination of the base or stock shows no malignancy." On the basis of this report, the patient was advised to have a total colectomy but at this time refused and in April, 1933 came back to the hospital with obstruction in the sigmoid and operation showed a generalized carcinomatosis from which he died four months later. No recurrence of the original growth was in evidence at any time.

This case is the typical picture which is seen so often where polyposis of the large bowel is present.

CASE 2. Private case, a female, aged fifty-three. This patient reported with thirty-six hours of acute intestinal obstruction with no previous story that could be obtained at any time other than vague digestive disturbances. Immediate operation showed what was apparently a carcinoma of the sigmoid and a cecostomy was done. At the second operation careful palpation showed not only the growth but about two inches below a mass which, at first, was thought to be feces but was found to be fixed so that the resection was made to include it. The pathologist's report was somewhat of a surprise as he said that the carcinoma was actually a diverticulitis and that the mass below was a polyp with a definite beginning malignancy. That patient has been followed routinely since without evidence of recurrence by x-ray or laboratory study.

This case is, of course, what would be called a "lucky break" for the patient and shows what is apparently a single polyp inasmuch as in four years no evidence of other polypi has developed. Nevertheless, I believe the burden of proof is on us to watch her and make sure that no further secondary cancer develops.

CASE 3. M. G. H., E. S., a man, aged fifty, came into the hospital in April, 1930 with symptoms extending back only a few weeks. Distress and indefinite indigestion were the main symptoms. There was a palpable mass in the right upper quadrant and x-ray showed a tumor of the hepatic flexure. At operation a resection was done and no metastases were found. The pathological report was adenocarcinoma and no involvement of lymph nodes, only a moderate rate of growth and a relatively low malignancy. He had no further symptoms until February, 1933 and he reëntered in April and at that time there was a growth in the upper end of the descending colon. Resection of the splenic flexure was done but this time there were metastases to the lymph nodes and when last seen four months ago he had a palpable liver and beginning jaundice. There was no evidence in the x-ray at any time either in 1930 or 1933 that there were polyps elsewhere in the colon and, at the last operation, careful palpation failed to show anything. This patient had not been followed between the two operations in the out-patient department.

I think this patient shows what is more apt to be the truth than the case previously noted; namely, that other polypi are present at the original operation but not palpable to the ordinary search. Had this case been followed in a routine fashion, it might well have been possible to recognize the secondary growth early enough to offer him still a good chance of permanent cure instead of which it has probably resulted fatally by this time.

CASE 4. A female, aged fifty-five, went to her doctor with a slight temperature of unknown origin and an increasing constipation, no localizing symptoms. An obstruction was demonstrated at the splenic flexure and in 1931 operation was done with a resection of the splenic flexure. The pathological report at that time was adenocarcinoma, grade 3. The patient did well and her check-ups were purely clinical until 1933 when it seemed to me that we must consider all of these cases as having potential malignancy elsewhere, and routine barium enemas were done as well as routine search for occult blood in the stool and rectal examinations. Everything went smoothly until the x-ray in September, 1934 which showed a slight filling defect in the low sigmoid. There was no other evidence of trouble, however, and she did not show positive guaiacs until the first of 1935. At that time she did have two or three positive guaiacs and another barium enema showed the same persisting suggestive area in the sigmoid. Accordingly, operation was done and an early carcinoma of the sigmoid was found. Because of the previous operation and the resulting tension, the large bowel from the middle of the transverse colon down to the lower sigmoid was removed and on section of the old anastomosis, an early recurrence was found in the wall. The lymph nodes were not invaded and it seems as though she had a better chance for cure than the usual secondary operation gives.

In this instance I had not reached the point that I now have where the suspicious x-ray should have resulted in earlier action.

With these cases as illustrations my thesis is simple. I believe that whenever a carcinoma of the large bowel is successfully operated on, that colon should be considered as having one or more starting points for later growths. Accordingly, I believe the following procedures should become routine: Before operation, the roentgenologist should report not only that there is an obstruction or a growth but also should, to the best of his ability, say whether there are other areas which suggest possible polypi. I believe that a proctoscopy should be done to see whether the rectum and low sigmoid will show polyps. At operation whenever it is possible, the surgeon should palpate carefully the whole area of the colon to see whether he can demonstrate other tumors. It is always difficult and oftentimes impossible to recognize small, soft, pedunculated papillomata so that this manoeuvre must not be considered sufficient to rule out future trouble. After the patient's recovery, these cases must be considered to have potential malignancy elsewhere in the large bowel until time and careful study have ruled it out. This study should mean a routine check-up at least every six months and should consist of the following: (1) A careful x-ray study by a roentgenologist competent to give reasonable hope of early recognition of trouble; (2) routine study of stools for occult blood. The patient should also be instructed to report any change in bowel habits that show

any tendency to persist or recur. It seems to me that we have here a clear indication to justify this bother and expense to the patient in contradistinction to innumerable cases of vague abdominal symptoms which when first seen suggest a functional background and where the patient would generally feel that it was an unfair hardship and expense to put him through a complete x-ray, proctoscopic, and laboratory check-up.

DISCUSSION

DR. LUCIUS C. KINGMAN, Providence, R. I.: *Mr. President*—Dr. Young has covered this whole matter very well. We have all seen cases such as he describes, and we are of the opinion that secondary operations in cancer of the big bowel are quite justified when we have symptoms pointing to a local condition. On secondary operation I have found obstruction due to cancer of small bowel, and of course that due to adhesive peritonitis is not uncommon.

As to how long a patient should be followed until we feel he is free from a chance of recurrence, I do not know. Probably all malignancy should be followed as long as the patients live or as long as you can persuade them to remain under observation.

On the hospital cases, I think the only practical method is a special clinic for malignancy. We cannot throw them into the general outpatient clinic or even the general follow-up clinic on service. The financial problem comes in, as Dr. Young mentioned, and at times we would be swamped if we followed these cases perhaps in the ideal way, which he has mentioned.

I should like to give a little word of warning about x-ray reports. I do not believe you can diagnose every case of malignancy of the big bowel by x-ray, even well advanced. I have been misled that way and in cases where they have been repeatedly x-rayed by experts in more than one town.

What he said about the complete examination of the big bowel at the time of operation is a very important thing, and probably most of us have not done that so well as we should. We go in and feel the growth, locate that, size it up. We are so interested in the job, we go ahead and do it. We forget there is a bowel below that may have polyps or implantations or what-not.

Dr. Young has told us several things that we should always bear in mind.

DR. FRANK H. LAHEY, Boston, Mass.: I have one thing to add and that is the value of contrast enemata. Lockhart-Mummery has just reported a series of cases of carcinoma of the rectum and colon, followed over a series of years, in which there was a high incidence of secondary carcinoma.

We have had this same experience, and everyone who deals with colons surgically is bound to have this same experience of there being coincident, other papillomata which may degenerate into malignancy. For that reason, I would urge strongly that everyone take advantage of the value of so-called contrast bismuth and air enemata for the demonstration of possible other coincident papillomata.

RETICULOCYTE RESPONSES IN THE PIGEON PRODUCED BY MATERIAL EFFECTIVE AND NONEFFECTIVE IN PERNICIOUS ANEMIA WITH DESCRIPTION OF THE HISTOLOGICALLY DIFFERENT REACTIONS OF THE BONE MARROW*

BY GULLI LINDH MULLER, M.D.†

IN the experimental work undertaken to obtain a biological test for the potency of material effective in pernicious anemia, one fundamental principle has hitherto not been emphasized, namely, that the reticulocyte response in the peripheral blood may represent at least two fundamentally divergent reactions of the bone marrow. This is not a question of theoretical interest only, but a vital problem, as a method must be found which will differentiate the response to the administration of the principle effective in pernicious anemia, and that of other substances closely associated with this principle, but without affecting in a desirable manner blood formation in the specific disease in man. That a reticulocyte response may be obtained in experimental animals, which bears no relation to the response obtained with substances potent in pernicious anemia, is the subject of this paper.

In 1927 shortly after the introduction of liver therapy in pernicious anemia, it was noted that the blood and the bone marrow of the pigeon were peculiarly susceptible to liver feeding[1]. In the autumn of 1929, comparatively pure sources of the principle effective in pernicious anemia were available, and it was shown, that material capable of causing a reticulocyte response with rapid blood regeneration in pernicious anemia also produced a reticulocyte response in healthy, grain-fed pigeons kept under *standard* laboratory conditions[2]. This has been confirmed by Edmunds and coworkers[3] and Peabody and Neale[4]. Wills[5] and Heimann, Connery and Goldwater[6] found that the reticulocyte percentage of all their birds showed large fluctuations and that no significant change occurred in the control animals and those which had liver extract. In a recent communication, Gurd[7] suggested that these differences may be due to the method of staining and counting reticulocytes. He found that the concentration of the brilliant cresyl blue exerted a marked influence on the number of reticulocytes in the pigeons' blood and that when a sufficiently strong solution was used some stained granular material was found in practically all the cells.

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The variations in experimental results obtained by various observers using different technics may be explained by a consideration of the sensitivity of the hematopoietic organs of the pigeons. Lack of food or inadequate diet may deplete the bone marrow even to the point of aplasia[1], and infection exerts a powerful influence[8]. Consequently in our comparative work on the effect of various substances on the hematopoietic organs of the pigeon over a period of years, the experimental conditions have been kept as identical as possible. Birds showing unexplainable variations in weight and in the blood during the control period were discarded. The pigeons were kept in small individual cages with space sufficient only for movement of the wings, and they were fed, and blood examinations made at the same time of day avoiding all undue excitement by rolling the animal in a towel. Great care was taken to avoid bleeding as in small animals the loss of several drops of blood may cause bone marrow changes. Also as was pointed out by Vaughan, Muller and Zetzel[2] more brilliant cresyl blue was used in the preparation of the blood smear than is necessary for mammalian blood by making thicker films using 0.5 per cent alcoholic solution of brilliant cresyl blue on *both* coverslips used for the smear. In counting the cells the fact was not emphasized in former communications that a few dust-like fine granules in the red blood cells were not considered as reticulum. Since the young cells of the birds' blood, as a rule, show heavy reticulation, the qualitative differences after the administration of certain substances were as striking as the quantitative change. In all our experiments the same technic was always employed, and the results obtained, therefore, may be considered significant. Reticulocyte responses accepted as positive were always in the form of a definite progressive rise rather than fluctuations back and forth between normal and higher levels. In addition, the response in the peripheral blood has been checked in most cases by microscopical examination of the bone marrow. Differences, therefore, found by some observers may be due to variations in the experimental conditions, and, as has been suggested by Gurd[7] to the staining and standard employed in counting the reticulocytes.

The megaloblastic character of the bone marrow in pernicious anemia and allied conditions is well established. Apparently, so far as is known, it is this type of bone marrow alone that responds to material effective in pernicious anemia. The quantitative and qualitative changes which occur in the bone marrow of patients receiving liver therapy have been described by Peabody[9].

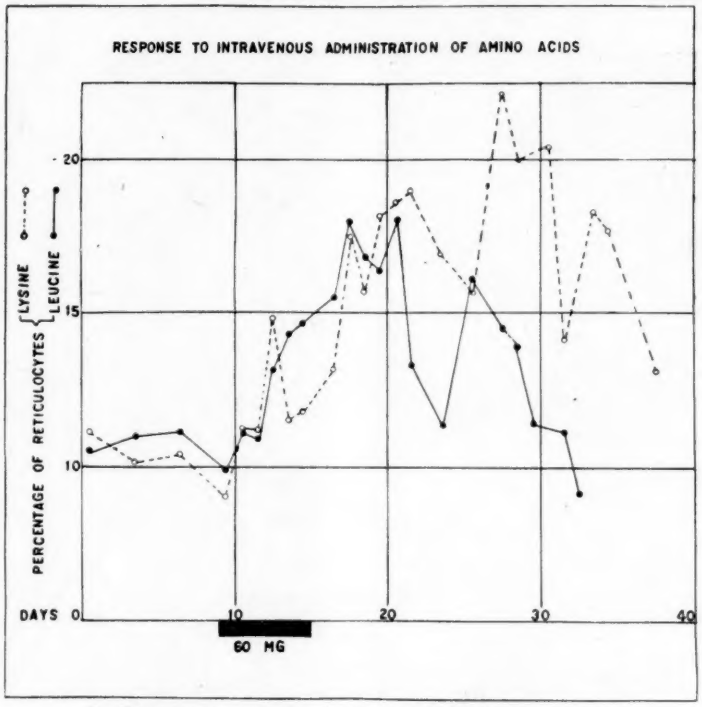
The bone marrow of the healthy adult pigeon likewise is characterized by an abundance of megaloblastic tissue. The administration of liver, or liver extract, potent for pernicious anemia, produced a reticulocyte response and changes in the bone marrow of the pigeon[1, 2, 8] similar to those observed by Peabody in man.

As no demonstrable effect had been obtained at that time in other laboratory animals with the principle effective in pernicious anemia, it was thought that this reticulocyte response in pigeons might furnish a convenient test for the potency of the principle effective in the disease. Further work demonstrated, however, that although most clinically inert fractions of liver elicited no reticulocyte response in the birds, one fraction, clinically tested and found ineffective, caused a reticulocyte response in pigeons. This fraction was a comparatively pure preparation of the amino-acid, leucine[2]; the preliminary work on leucine has been repeated and verified.

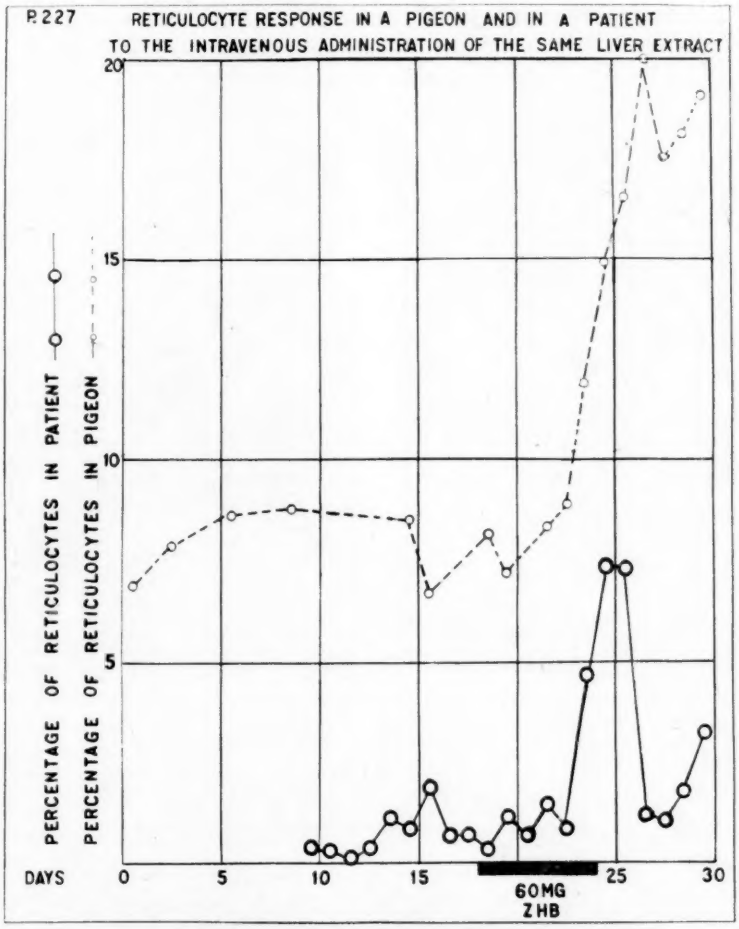
To find out whether commercial preparations of pure amino-acids, which are known constituents of red blood cells, produced a reticulocyte response in the peripheral blood of pigeons, three other amino-acids, lysine, arginine, and histidine have been tested. The experimental methods were identical with those previously employed[2]. Ten milligrams, and in some instances 20 milligrams of the amino-acid was administered intravenously on six consecutive days to several birds.

In chart 1 the reticulocyte responses to lysine and leucine are illustrated and in chart II the response to a comparatively pure fraction of liver extract injected intravenously. The reticulocyte reaction to the principle effective in pernicious anemia usually was a moderate one, only occasionally exceeding 20 per cent. Similar reticulocyte responses were obtained with both leucine and lysine. When larger doses of lysine were employed (20 mg.) there was an outpouring of reticulocytes in the peripheral blood of the pigeon in some instances reaching values of 30 to 35 per cent. The two other amino-acids tested, arginine and histidine, gave indefinite or no reticulocyte responses.

An explanation for the rise of reticulocytes in the peripheral blood of pigeons to substances which are not effective in pernicious anemia was found by the examination of the bone marrow. As has been pointed out previously[1, 8] the *radial* bone marrow in the normal pigeon has been found to be mainly fatty with some cellularity at the edges. The *femoral* marrow on the other hand was found to be hyperplastic and contains red blood cell islands consisting of a mixture of cells ranging from megaloblasts to mature red blood cells with a considerable proportion of the cells in the megaloblastic or early erythroblastic stage. Often mitotic figures are present. Occasionally the radial marrow is hyperplastic which occurs when the femoral marrow, for some unknown reason, has been replaced by cancellous bone. Hyperplasia, therefore, of the



I. Reticulocyte responses in the peripheral blood of pigeons to the intravenous injection of 10 mg. of lysine and leucine on six consecutive days.



II. Reticulocyte response in a pigeon following the injection of 10 mg. of an effective liver preparation on six consecutive days and the reticulocyte response in a patient with pernicious anemia who received a total of 1.6 grams of the same liver preparation. The patient's red cell level was 2,700,000 per cmm.

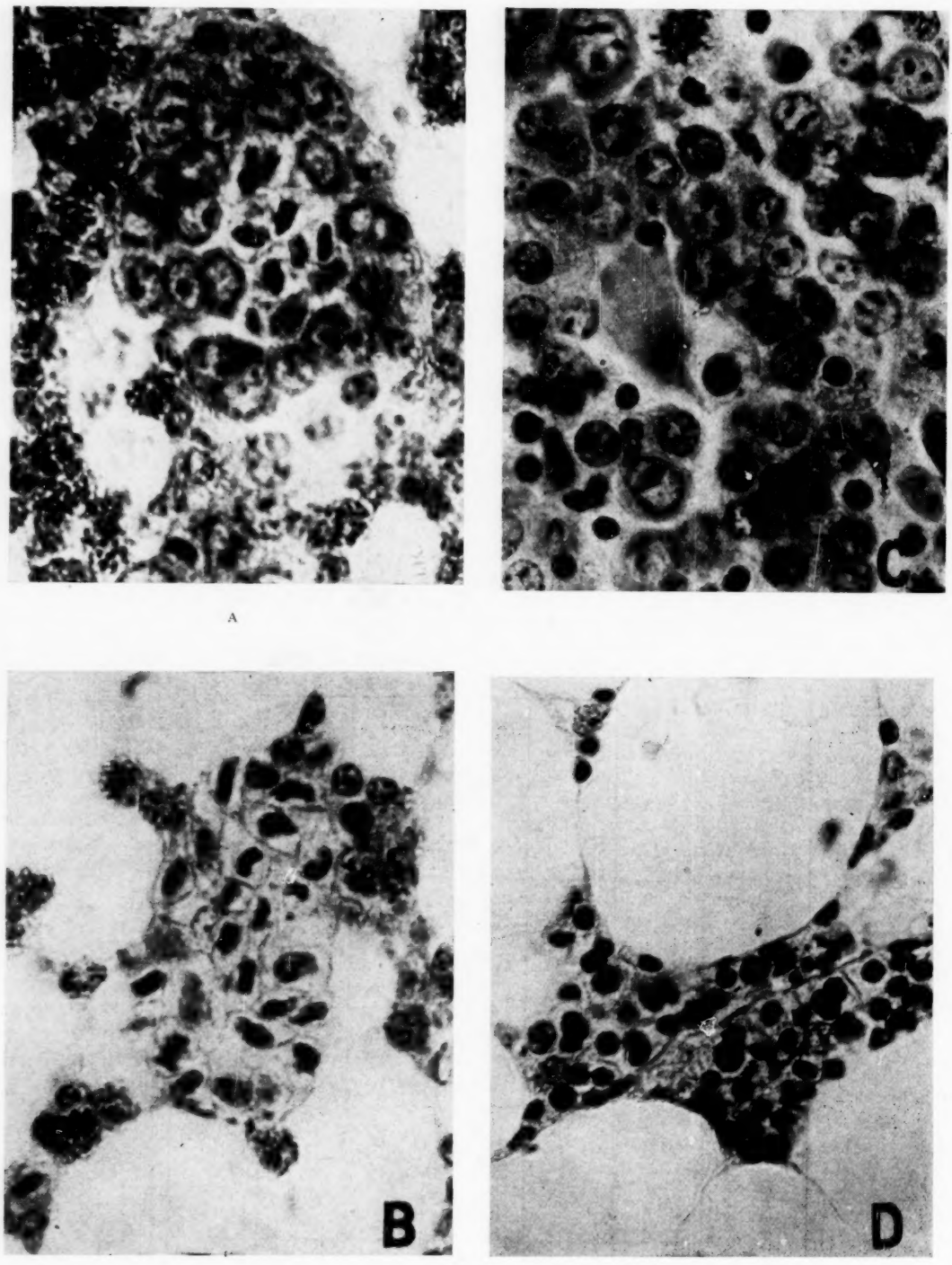


FIGURE 1. Bone marrow from pigeons and a patient with pernicious anemia before and after the administration of liver extract.

a. Normal *femoral* marrow of pigeon. Note the megaloblasts at the periphery of the island of erythrocytes and the adult cells in the center. ×1500.
b. Femoral bone marrow of a pigeon after the administration of liver extract. The island consists of practically mature cells. ×1500.
c. Tibial bone marrow from a patient with pernicious anemia in relapse. ×1000.
d. Tibial bone marrow from the same patient as (c) in a remission produced by the administration of principles effective in pernicious anemia. ×750.

radial marrow, as a rule, indicates growth and thus extension of the blood-forming tissue.

In fig. 1a the appearance of the normal femoral bone marrow of a pigeon under the standard laboratory conditions is illustrated, and in fig. 1b the femoral bone marrow after the administration of "liver extract". A marked change in the erythrocytic centers is discernible. The marrow obtained after the administration of liver extract, at a time when the peak of the reticulocyte response had passed, showed a qualitative change of the red blood cells. The erythrocytic centers were small and shrunken and consisted mainly of cells in the late erythroblastic and normoblastic stage. Quantitatively there appeared to be a decrease of erythropoietic tissue. Thus the outpouring of the reticulocytes occurred without microscopic signs of activity and without extension of erythropoietic tissue into the radial marrow.

The similarity of this response in the birds' bone marrow to the one obtained in patients with pernicious anemia is striking. This is illustrated in figs. 1c and d. The former shows a section of the tibial bone marrow of a patient with pernicious anemia in relapse and the latter the marrow from the same patient, both obtained by biopsy, after the administration of substances effective in pernicious anemia[9].

In figs. 2a and b is shown the *radial* marrow of a pigeon injected intravenously with lysine. The extension of erythrocytic centers and the hyperplasia of this normally practically fatty marrow and the activity, as evidenced by mitotic figures, is unmistakable. The reticulocyte response to lysine, therefore, and other substances examined giving the same bone marrow reaction, is thus easily explainable. New formation of erythrocytes in the femoral bone marrow, and more especially, extension to the radial marrow accounted for the reticulocyte response both qualitatively and quantitatively. The reticulocyte response to the principle effective in pernicious anemia, however, seemed to be commensurable with the amount of megaloblastic tissue present and there was no stimulation of the bone marrow, if with stimulation we postulate new formation of megaloblasts and extension of the erythropoietic tissues.

From the above, one is justified in concluding that a reticulocyte response in the peripheral blood may depend upon at least two diametrically opposite reactions. In one a megaloblastic marrow, as is found in patients with pernicious anemia and allied conditions and in the normal pigeon, is essential. This type of marrow is influenced in a specific manner by the principle effective in pernicious anemia. The other type

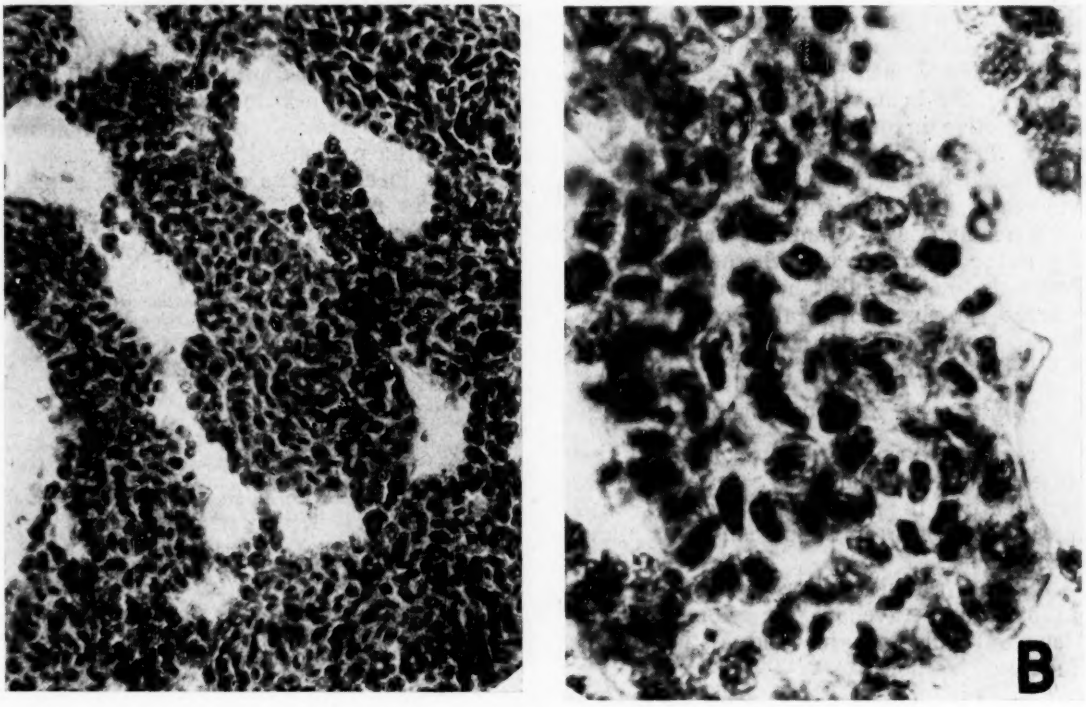


FIGURE 2. *Radial* bone marrow of pigeon injected intravenously with 20 mg. of lysine for ten consecutive days and killed sixteen days after the first injection.

a. Hyperplasia of erythroblastic tissue in a normally fatty marrow. ×500.
b. Large erythroblastic center from fig. a, showing megaloblasts and several mitotic figures. ×1500.

of reticulocyte response produced by leucine and lysine in this study, is caused by a stimulation and proliferation of red blood cells and an extension of blood-forming tissue. Qualitatively and quantitatively the reticulocyte response in the pigeon to substances causing a stimulation and extension of erythropoietic tissue cannot be differentiated from that due to the specific effect of "liver extract". It is obvious, therefore, that this reaction alone cannot be used as a measure of the potency of unknown principles as to their effectiveness in pernicious anemia. However, in the hands of the experienced observer, the reticulocyte response in the peripheral blood of the pigeon, correlated with the histological appearance of the bone marrow might become a valuable, although not ideal, biological indicator of the hematopoietic potency of substances effective in pernicious anemia. Recently a test for the potency of liver extract has been proposed by Jacobson[10], namely, the reticulocyte response in particularly selected guinea pigs. A mammalian test-animal has many advantages over the avian, but whether the guinea pig responds to other substances with a reticulocyte increase in the peripheral blood and hyperplasia and extension of the erythropoietic tissue has not been reported.

SUMMARY AND CONCLUSION

1. Previous work has been confirmed that the injection of comparatively pure liver extracts effective in pernicious anemia causes reticulocyte responses in the pigeon under standard conditions.

2. Similar reticulocyte reactions were obtained by the intravenous injection of lysine and leucine, while arginine and histidine gave but indefinite responses.

3. These reticulocyte responses in the peripheral blood were accompanied by two histologically different reactions of the bone marrow.

a. After the administration of liver extract the megaloblasts of the bone marrow were transformed to more adult red blood cells. This occurs also in the bone marrow of pernicious anemia patients after liver therapy.

b. After the administration of lysine and leucine the bone marrow showed an increase and extension of erythroblastic tissue. Numerous megaloblasts and many mitotic figures were present.

4. A reticulocyte response in the peripheral blood, therefore, may be due either to a principle effective in pernicious anemia acting on a megaloblastic marrow, or to other substances such as lysine or leucine, which produce a growth and thus extension of erythrogenic centers. This dual reaction is a factor to be considered in any work undertaken for a biological test of the principle effective in pernicious anemia, as the peripheral reticulocyte response may be produced by two diametrically opposite bone marrow reactions.

REFERENCES

1. Muller, G. L.: Experimental bone marrow reactions; influence of liver and meat diets on bone marrow and regeneration of red blood cells and hemoglobin. Am. J. Physiol. **82:** 269 (Oct.) 1927.
2. Vaughan, J. M.; Muller, G. L., and Zetzel, L.: The response of grain-fed pigeons to substances effective in pernicious anaemia. Brit. J. Exper. Path. **11:** 456 (Dec.) 1930.
3. Edmunds, C. W.; Brueckner, H. H., and Fritzell, A. I.: On laboratory test for liver extract. J. Am. Pharm. A. **22:** 91 (Feb.) 1933.
4. Peabody, W. A., and Neale, R. C.: The pigeon as a hematopoietic test animal. J. Am. Pharm. A. **22:** 1231 (Dec.) 1933.
5. Wills, L.: Spontaneous fluctuations in reticulocyte count in pigeon's blood. Brit. J. Exper. Path. **13:** 172 (April) 1932.
6. Heimann, H.; Connery, J. E., and Goldwater, L. J.: Lack of effect of liver treatment on the circulating reticulocytes in the pigeon. Am. J. M. Sc. **188:** 343 (Sept.) 1934.
7. Gurd, M. R.: The use of grain-fed pigeons in the biological assay of liver preparations. Quart. J. Pharm. & Pharmacol. **8:** 39 (Jan.-March) 1935.
8. Muller, G. L.: Influence of liver extract and acute infection on reticulocytes and bone marrow of pigeons. Arch. Path. **14:** 774 (Dec.) 1932.
9. Peabody, F. W.: The pathology of the bone marrow in pernicious anemia. Am. J. Path. **3:** 179 (May) 1927.
10. Jacobson, B. M.: Response of normal guinea pig to administration of liver extracts. Science **80:** 211 (Aug.) 1934.

SPINAL ANESTHESIA*

Agents, Methods and Indications

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WITHIN the past few years there have been added to the field of spinal anesthesia new methods and agents. This paper is offered as a review of the subject with an attempt to consider and contrast the more important agents, methods and the theories underlying their usage.

AGENTS

In considering agents employed in any field of usefulness to accomplish a given purpose it is logical to investigate first their relative toxicity.

Some may argue that the toxicity of an agent employed in spinal anesthesia is not important since the dose necessary to accomplish a therapeutic result is so small that the danger from toxic absorption into the blood stream is negligible. Witness the statement of Babcock[1] who speaks of the successful use of cocaine intraspinally in the General Hospital of Mexico City. It is his feeling that "the danger lies more in the technique of the user than in the drug."

Nevertheless, if a difference in toxicity between agents does exist, it is important that we know and benefit by it. Toxicity of an agent

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employed in spinal anesthesia may manifest itself by absorption from the subarachnoid space with action on the body as a whole or secondly by its action on the structures with which it comes in direct contact.

First, its action on the body as a whole: A rather complete study of all available English literature on the subject failed to produce an investigation on the relative action of the several agents employed in spinal anesthesia determined through this method.

Marvin[27], in considering the present status of various spinal anesthetics, quotes the work of Nowak in determining the comparative fatal intravenous dose of the various anesthetics on cats. Nowak[33], in a recent communication, states his conclusions are in this regard unchanged.

Another investigation of the comparative fatal intravenous dose of the various agents is that of Wiedhopf[45]. This work was done on rabbits.

Their work is summarized in table 1. In the

TABLE 1

COMPARATIVE FATAL INTRAVENOUS DOSE

	Avge. Fatal Dose Mgm./kgm.	Ratio (Compared to Procaine)	Dose Ratio	Corrected Toxicity Ratio (Compared to Procaine)
Cats (Nowak)				
Procaine	49.6	1.0	1.0	1.0
Metycaine	28.8	1.7	0.8	1.36
Pantocain	8.6	5.8	0.1	0.58
Nupercaine	3.5	14.2	0.05	0.71
Rabbits (Wiedhopf)				
Procaine	57.5	1.0	1.0	1.0
Pantocain	8.0	7.18	0.1	0.71
Nupercaine	3.0	19.16	0.05	0.958

first column we see the average dose in milligrams per kilogram necessary to kill the animal. In the second column we note the toxic ratio of the various agents, compared with procaine. Thus, giving procaine unity, we find, in Nowak's work, that metycaine is 1.7 times as toxic as procaine, pantocain 5.8 and nupercaine 14.2 as toxic. In Wiedhopf's work metycaine was not considered. Here we find pantocain 7.18 times and nupercaine 19.16 times as toxic as procaine.

These studies up to this point show the relative absolute toxicities of the drugs. To stop here would be unfair to the different agents and would not be truthful. To approach the truth a bit closer it would be necessary to consider another factor—that of relative dosage. Marvin[27] gives us the relative dosage of the agents. Table 2. To his list of relative dosage

TABLE 2

RELATIVE DOSAGE
(Marvin)

	Mgm.	Ratio
Procaine	200	1.0
Metycaine	160	0.8
Pantocain	20	0.1
Nupercaine	10	0.05

I have added the dose ratio. Giving procaine unity we find that metycaine is effective in 0.8 the dose, pantocain 0.1 and nupercaine 0.05 the dose of procaine. Transposing this dose ratio to table 1 and correcting the ratio of absolute toxicity we arrive at a corrected toxicity ratio. Here we find, in Nowak's work, that metycaine is 1.36 times as toxic as procaine, pantocain 0.58 and nupercaine 0.71 times as toxic as procaine. In Wiedhopf's work we see that pantocain is 0.71 and nupercaine 0.958 times as toxic as procaine. Here I wish to stress a very important factor. The work here quoted was not done on human beings. It is true that animal investigation is very helpful but we must not lose sight of the fact that animals are prone to react to drugs differently than is the human being.

Another reason why we should not accept this corrected ratio at its face value is the fact that the final ratio arrived at is dependent to a large extent on the dose ratio of the various agents. There exist in the minds of many anesthetists marked disagreements as to the relative dosage of the various drugs. Were we to accept the finding of Israëls and MacDonald[10] that in intraspinal injection in the cat nupercaine is as effective as procaine in 1/40 of the dose instead of Marvin's feeling that it is as effective as procaine in 1/20 the dose, we can see the difference it would make in the corrected toxicity. As far as I have been able to determine no one has subjected a human being to the tests necessary to determine the relative dosage of the agents considered in preventing the passage of impulse in an exposed motor or sensory nerve.

In considering the toxicity of the agents we should not depend entirely on laboratory findings. The clinical aspect is equally important. Lundy[23], in speaking of the advantages and disadvantages of the various anesthetic agents, divides them into two groups from the standpoint of immediate results:

1. Those in which there is little if any margin of safety between failure of the respiration and failure of the heart.
2. Those in which there is a wide margin of safety between failure of respiration and failure of the heart.

In the first group he places nupercaine, in the second procaine. He did not class metycaine or pantocain. On at least two other occasions he expressed himself as to the comparative toxicity of the agents. On one occasion, speaking of various anesthetic agents, he said[22], ". . . no safer one than procaine hydrochloride", and on the other[21], "Since procaine hydrochloride is the safest. . . ."

Essex and Lundy[9] feel that the same dose of the same drug assumes different toxicities under different conditions. They feel that the concentration of the drug is important, as well as the number of milligrams of the drug used. A given number of milligrams of a given drug tends to be less toxic in high than in low dilution.

Recent investigation into the irritative action of agents on the spinal cord or nerve roots has been under way. Lindemulder[20] showed neurological changes after apothesin spinal anesthesia. In two cases he was able to show structural changes in the spinal cord. In one case there was extensive degeneration of the fibres in some small nerve roots with a loss of myelin sheaths. This is a severe charge to lay at the feet of this drug. In Lindemulder's cases it is important to know that 1.5 cc. of apothesin solution was injected with no barbotage and followed by immediate Trendelenburg position. The question to ask one's self is—would a solution of lower concentration have caused this? Or, had barbotage been employed with resultant dilution would this damage have been as likely to occur?

Nonne and Demme[32] report two cases of myelitis following the use of tutocaine intraspinally.

Lundy and others[24], in experiments on dogs, were able to produce permanent and fatal paralysis by intraspinal injection of procaine. This they were able to do only by using tremendous doses. Thus, they were able to show damage by injection of 5 cc. of a 20 per cent solution of procaine, 1000 milligrams. No permanent paralysis followed the administration of 5 cc. of a 17.5 per cent solution or of 5 cc. of any weaker solution.

Davis and others[7] studying all the present agents found inflammatory reaction in the leptomeninges, degenerative changes in the fibre tracts, etc. In dogs allowed to live ninety days the reactions were not pronounced. They feel that the reactions were transitory. They did no work on the human being.

Koster[18], working on frogs, was able to show cord reaction following intraspinal injection of procaine. These changes he found were transitory, beginning to regress in six hours and disappearing entirely in twenty-four hours after the intraspinal injection. He examined the spinal cords of human beings who died at varying intervals following spinal anesthesia from other causes and found no histologic changes from the normal. These cords were examined 22, 36, 96, 144, 192 and 816 hours after the anesthesia.

Unless added cases of cord damage following spinal anesthesia present themselves I feel it safe to say that the possibility of cord or nerve root damage following the use of the present agents is very unlikely.

In addition to difference in toxicity and range of dosage, the agents vary also as to their speed of activity. Woodbridge[48] feels that metycaine produces anesthesia a little more quickly than does procaine. Most observers feel that the action of nupercaine is considerably slower than that of procaine. The duration of action of the drugs is given as follows[27]: procaine, one hour; metycaine, ninety minutes; pantocaine, two hours and nupercaine, three hours. Most observers agree to the relative duration as above given, with some differences as to duration of action.

All the agents produce a fall in blood pressure if administered in sufficient dosage and if the anesthesia is high enough. Most workers with pantocain and nupercaine feel that the fall in blood pressure with these agents is less than that which may occur following the use of procaine.

All the agents may depress respiration. McCuskey[30] states that pantocain seems to have a more rapid action on the respiratory center than procaine, and, due to its longer action as an anesthetic, extreme care should be used to avoid respiratory accidents. Jones[12], employing a hypobaric solution of nupercaine, feels that paralysis of the intercostals may be more marked than with procaine because of the predominant anterior root action of this hypobaric solution. Here, too, the action is apt to be very much prolonged and care is here also important.

The relative actions of metycaine and procaine on respiration I do not know. My impression is that they do not differ markedly.

As to the stability of these drugs, procaine seems to be very resistant to heat and storage. It is capable of resisting heat up to 120 degrees Centigrade without deterioration. It is very soluble, forming a clear stable solution with quantities as low as its own weight of spinal fluid. Metycaine in solution does not deteriorate by boiling and is readily soluble in water[29]. In solution it seems to have an antiseptic property. High temperatures such as prolonged boiling or storage near a radiator, may cause decomposition of pantocain[26]. It can, however, be easily sterilized. Crystals of nupercaine are neither soluble in spinal fluid nor in saline solution. Solutions of nupercaine, as sold by the manufacturer, are buffered solutions. The 1:200 solution contains, in addition to nupercaine, NaCl, Na_2HPO_4 and NaH_2PO_4. In neutral

or alkaline state precipitation may occur. In the weak solutions of nupercaine, 1:1000, 1:1500 and 1:2000, it is necessary to acidify the solution by the addition of a few drops of dilute hydrochloric acid. Romberger, in combining nupercaine with procaine, formerly employed the 1:200 buffered solution. He is, at present[37], using a 1 per cent solution of nupercaine in buffered saline to which 0.5 per cent lactic acid has been added, in order to overcome any possibility of precipitating the nupercaine base when the solution is injected into a column of spinal fluid. The manufacturers are not putting up this combined solution claiming it is not stable.

PREOPERATIVE MEDICATION

The use of morphine or pantopon with either atropine or scopolamine as a preoperative medication is general. Occasionally moderate doses of the shorter acting barbiturates are employed. CoTui[8] has shown that "small but effective doses of morphin, morphin-scopolamine, and morphin-avertin given prior to cisternal injections do not cause a determinable decrease in the minimum lethal dose of the drug while larger doses do, and that sodium amytal narcosis, whether light or profound, produces a marked reduction". In this latter regard it is interesting to note that Jarman[11] in addition to administering intravenously pantopon gr. 2/3, scopolamine gr. 1/150 adds a small dose of either evipal, nembutal or pernocton by the same route.

EPHEDRINE

The use of ephedrine as a vasopressor in preventing the fall in blood pressure is almost universal. There are a few dissenting voices to be heard protesting its use. Those employing it do so almost routinely on all patients for intra-abdominal surgery, with probably marked hypertension as the only contraindication. The occurrence of cardiac irregularity, extrasystoles and cardiac dilation they never think of blaming on the ephedrine, though certainly this drug may be responsible for such action[31, 35, 39]. Labat does not counsel its use. Vehrs[44] feels that pressor agents in general are detrimental to success and that they should not be used as a routine measure. The objectors, it must be noted, are in the minority. CoTui[6] feels that ephedrine is of definite value. This he has demonstrated in laboratory experiments on animals. He concludes that the use of ephedrine is considerably more efficacious than either saline infusion or transfusion.

Meeker and McCreary[29] use 75 mgm., with metycaine as the agent. Sise[40] recommends for operation below the abdomen "very little" ephedrine. For abdominal operations with patients in Trendelenburg position he recommends 50 mgm. and with patients level 75-100 mgm. Tables 3, 4 and 5 show the position of other workers in regard to ephedrine with dosages for the various heights of anesthesia. I wish to state at this point that the tables 3, 4 and 5 repre-

TABLE 3

PROCAINE TECHNIQUES

	Perineum	Lower Abd.	Mid Abd.	Upper Abd.	Remarks
Labat					
Dose in mgm.	100	120	120	150	
Ephedrine in mgm.	0	0	0	0	
Dilution in cc.	3	5	5	5	Approximate.
Trendelenburg	+	+	+	+	Immediate.
Site of Puncture	L3	L2	L1	D12	
Evans					
Dose in mgm.	60	100	120	120	
Ephedrine in mgm.	0	0	0	0	Adrenalin intramuscularly.
Dilution in cc.	3	5-9	7-9	8-11	
Trendelenburg	0	0	0	0	Only if vasomotor collapse or if anesthesia too high.
Site of Puncture	L5	L3	L3	L2	
Stout					
Dose in mgm.	100	150	150	200	Speed of inject. Perineum 5 secs.
Ephedrine in mgm.	0	30	50	80	Lower abd. 7 secs.
Dilution in cc.	1	1.5	2	2.5	Mid abd. 10 secs.
Trendelenburg	0	0	0	0	Upper abd. 12 secs.
Site of Puncture	L2-L3	L2-L3	L2-L3	L2-L3	Ten min. after inject. any position.
Vehrs					
Dose in mgm.	85	110	175	225	
Ephedrine in mgm.	0	0	0	0	
Dilution in cc.	1	2	2	4	
Trendelenburg	+	+	+	+	In mid and upper abd. moderate to deep Trendelenburg is delayed.
Site of Puncture	L4	L3	L3	L2	
Rovenstine					
Dose in mgm.	50-75 plus .5-.75 cc. 10% Dextrose				
Ephedrine in mgm.	0				
Dilution in cc.	1.5-2				For transurethral surgery. Patient in lithotomy, head and shoulders elevated.
Trendelenburg	0				
Site of Puncture	L4-L5				

TABLE 4

NUPERCAINE TECHNIQUES

	Perineum	Lower Abd.	Mid Abd.	Upper Abd.	Remarks
Keyes 1:200					
Dose in mgm.	10	10	10	10	
Ephedrine in mgm.	100	100	100	100	
Dilution in cc.	2	5	8	10	
Trendelenburg	+	+	+	+	Immediate.
Site of Puncture	L3	L3	L3	L3	Pt. upright.
Jones 1:1500					
Dose in mgm.	4	6.6	8	10-12	
Ephedrine in mgm.	60	70	80	90	Approximate.
Dilution in cc.	6	10	12	15-18	After injection patient prone 5 min.
Trendelenburg	+	+	+	+	
Site of Puncture	L4	L1	L1	L1	Lateral position.
Ehrlich 1:1500					
Dose in mgm.	3.3-6.6	5.3-6.6	6.6	6.6-8	
Ephedrine in mgm.	50	50	50	50	
Dilution in cc.	5-10	8-10	10	10-12	
Trendelenburg	+	+	+	+	Positions as above.
Site of Puncture	L3	L2	L2	L1	
Wilson 1:1500					
Dose in mgm.	6.6	6.6	8	10	Patient upright
Ephedrine in mgm.	50	50	50	50	for low anesthesia 20 secs.
Dilution in cc.	10	10	12	15	mid anesthesia 30 secs.
Trendelenburg	+	+	+	+	upper anesthesia 40 secs
Site of Puncture	L3	L3	L3	L3	
Romberger Nupercaine-Procaine combined					
Dose in mgm. { Nupercaine	6-8	8-10-12			
Dose in mgm. { Procaine	75-100	120-240			All abdominal cases blocked to nipple line.
Ephedrine in mgm.	50	75			
Dilution in cc.	0.5-1	2-2.5			
Trendelenburg	+	+			Delay, then use as needed.
Site of Puncture	L4	L2-L1			

TABLE 5

METYCAINE TECHNIQUE

	Perineum	Lower Abd.	Mid Abd.	Upper Abd.	Remarks
Woodbridge					
Dose in mgm.	40-80	120	140	180	Average.
Ephedrine in mgm.	25	50	50	50	
Dilution in cc.	2	4-5	5	5	
Trendelenburg	0	Trendelenburg for height of anesthesia, then flat.			
Site of Puncture	L4	L3	L3	L3	

PANTOCAIN TECHNIQUE

	Perineum	Lower Abd.	Mid Abd.	Upper Abd.	Remarks
McCuskey					
Dose in mgm.	8	12	12	15	
Ephedrine in mgm.	25	25	25	25	
Dilution in cc.	1.6	2.4	2.4	3	Approximate.
Trendelenburg	+	+	+	+	
Site of Puncture	"One level higher than with procaine"				

sent dosages and other factors with a good risk adult of average size, weight and physical condition as the patient. The factors, as given, are average. Anesthetic judgment comes into play in using these tables. The patient's physical condition, type of surgery, speed of operator and very many other conditions may alter any and all the factors given. Some of the factors quoted are the average of two given by the worker.

METHODS

The methods employed in spinal anesthesia are essentially attempts to place an anesthetic fluid subdurally about nerve roots. A knowledge of the anatomical relationship between the lower spinal vertebrae and segments of the cord is necessary.

A solution deposited at one point subdurally may be transferred in part to another location about the cord by one or usually by a combination of the following factors:

1. *Diffusion.* Diffusion may be controlled by—

a. Dosage. A drug in solution placed intraspinally will very soon begin to lose its original concentration, becoming diluted with the medium in which it finds itself. This increasing dilution will occur in all directions. Any nerves exposed become affected in proportion to the concentration of the drug surrounding it. Nerves at a distant point may not receive a solution sufficiently strong to alter their action. Thus, the greater the original concentration the more widely will the action of the drug be effective. This factor may be spoken of as molecular diffusion.

b. Volume. Of two volumes of an anesthetic solution, of like strength, the larger will occupy a greater area within the subarachnoid space and the smaller a less. In each case they involve an area in proportion to the amount of fluid injected. Whether we begin the injection with 2 cc. of a solution and by barbotage increase it to eight or whether our original dose is immediately dissolved in eight cc. and injected with no barbotage, it is an attempt to deliver the drug to a site within the subarachnoid space distant to the point of needle entrance, by diffusion. The difference is slight but it is there. Employing barbotage, we are continually injecting solutions of diminishing concentration, whereas, in the absence of barbotage we are injecting a solution of definite characteristics, the nature of which we know beforehand. Because of the difference in the characteristics of the solutions, varying results may be expected.

2. *Site of Injection.* As readily seen by varying the level at which the anesthetic solution enters the dural canal one can approach a point closer or more distant to the nerve roots to be blocked.

3. *Speed of Injection.* Likewise readily seen is the fact that a solution forcibly injected would reach a point farther from the site of needle entrance than had the solution been gently introduced.

4. *Specific Gravity.* Considerable has been said relative to the importance that specific gravity plays in determining the height of anesthesia. Table 6 is a list of the different specific gravities of spinal fluid as determined by various workers. This is a table from an article by Jones[12]. It is very important to note the wide variation that occurs, from 1.001 to 1.010. Others give the specific gravity of spinal fluid as from 1.0012 to 1.0060[26] and from 1.006 to 1.008[16].

TABLE 6

SPECIFIC GRAVITY OF SPINAL FLUID

Hoppe	1001-1005
Haliburton	1007-1008
Kazka	1002-1008
Zdareck	1007
Polanyi	1005-1007
Mestrezat	1007
Nawratzat	1008
Penda	1007-1010
Borelli and Data	1007-1009
Williamson	1005-1009
Hammerstein	1007-1008
Eskuchen	1006-1007
Levinson	1003-1007

TABLE 7

SPECIFIC GRAVITY OF AGENTS USED IN SPINAL ANESTHESIA

	Specific Gravity
Procaine	
1:10 in spinal fluid	1.025
1:20 (200 mgm. in 4 cc.)	1.013
1:40 (200 mgm. in 8 cc.)	1.008
Metycaine	
1:20	1.020
1:10	1.036
Pantocaine	
1:100 in saline	1.0068
1:400 in saline	1.002
Nupercaine	
1:200 0.5% saline	1.0046
1:1500 0.5% saline	1.0030
1:1500 0.9% saline	1.0061
1:1500 in water	1.00005
10% Procaine in 1:200 Nupercaine	1.018

Table 7 lists the specific gravities of the various anesthetic solutions. The items in this table were taken from various sources[12, 13, 28, 30, 36, 43, 45]. To use the terms employed by Jones to represent solutions with a specific gravity heavier than, the same as, or lighter than spinal fluid-hyperbaric, isobaric or hypobaric—and, considering the extremes in specific gravity which spinal fluid might have, we find that the same agent in the same dilution may at different times assume any of the above relationships to spinal fluid.

Thus pantocain, 1 per cent in saline, with a specific gravity of 1.0068 may be hyperbaric to a spinal fluid of less specific gravity, isobaric to a fluid of like specific gravity and hypobaric to one of greater gravity.

Solutions to be always considered hyperbaric

would have to have a specific gravity greater than 1.010. In this classification fall most of the procaine or metycaine solutions. Solutions to be consistently hypobaric would have to have a specific gravity of less than 1.001. Under this heading would be the 1:1500 solution of nupercaine in water.

Romberger[36] employs a small bulk concentrated solution of high specific gravity. He feels that a greater degree of control is thus obtained. It is his feeling that the rate of diffusion is fixed and certain and that gravitation can be regulated by the level or plane of the spine. Jones[12] attempts to create within the dura a long or short column of anesthetic fluid depending on the height of anesthesia desired. To this end he employs a 1:1500 solution of nupercaine with a specific gravity of 1.003. This "hypobaric" solution if introduced into a spinal canal the fluid of which has a specific gravity of 1.001 becomes actually a hyperbaric solution. Under this condition, employing Trendelenburg position, an anesthesia higher than that expected will occur.

It is difficult for many to believe that a small difference in specific gravity between the agent and spinal fluid is important, especially since the agent and spinal fluid are so readily soluble in one another.

The higher area of sensory anesthesia over motor which occurs under spinal anesthesia we have been led to believe is a result of the greater selective affinity of sensory over motor nerves for local anesthetics, particularly in weak solutions. Jones[12] offers an added explanation for the phenomenon. He reasons that to depend on gravity to give proper anesthesia and muscular relaxation, employing a hyperbaric solution, results in higher sensory anesthesia than motor since the posterior roots are bathed over a larger area than are the anterior. (Figure 1A.)

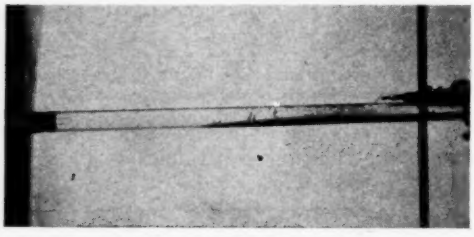

FIG. 1A. Dye represents a solution of greater specific gravity than spinal fluid.

In employing a hypobaric solution the opposite occurs—a higher motor than sensory anesthesia. This is the result of a more widespread anterior root action. (Fig. 1B.) Thus in using solutions of lower specific gravity than spinal fluid paralysis of intercostal nerves may be more marked than with the other type of solution.

5. *Position of Patient.* Changing the tilt of the table as employed in the various techniques of spinal anesthesia, is for one or both of the following reasons:

a. To place the anesthetic solution about some desired segments of the cord.

b. To prevent or treat certain dangerous complications which may arise as the result of the fall in blood pressure accompanying the anesthesia.

Most workers using agents with a specific gravity higher than that of spinal fluid employ the Trendelenburg position at some point in their technique to increase the height of anesthesia. Those dealing with a solution of lighter

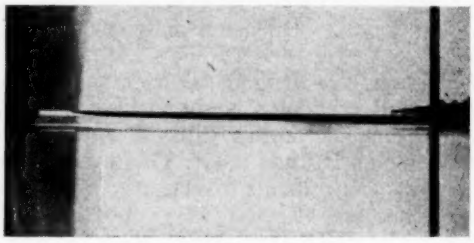

FIG. 1B. Dye represents a solution of lower specific gravity than spinal fluid.

specific gravity place their patients in the same position to keep the solution low down in the canal. Occasionally, workers with a heavy solution will raise the head of the table to maintain the agent in the most dependent portion of the dural space. Thus Romberger[36] administering a concentrated procaine-nupercaine mixture uses Fowler's position to control height. He employs the Trendelenburg position later on as needed, but he warns that there is danger in the too early use of the head down position since nupercaine fixes slowly and in combination with procaine is heavier than spinal fluid. McCuskey also delays, for a period of fifteen to twenty minutes, assumption of the Trendelenburg position after the introduction of a pantocain-procaine combination, for the same reason as does Romberger. Rovenstine[38] in dealing with a procaine-dextrose solution, particularly in transurethral prostatic resections, recommends that the head and shoulders be elevated to maintain a low anesthesia. Etherington-Wilson[46] employs the sitting position for varying intervals following injection of a 1:1500 solution of nupercaine, allowing for rise of the agent in the canal.

Jones[13] feels that application of the Trendelenburg position to give height of anesthesia results not in moving a dose in high concentration to the most dependent part but to advance an ever-decreasing concentration over a distance which will be proportional to the amount of drug injected. Woodbridge[47] administering metycaine employs Trendelenburg in preference to barbotage to give height of anesthesia.

CoTui[4, 6] in spinal anesthesia on dogs showed that in the head down position it takes less than one-half the dose to kill an animal than in the horizontal position. In the head up position it takes even more to kill the animal. The

above experiments were with procaine dissolved in 3 cc. of distilled water and injected in the first lumbar interspace. In the horizontal position it takes over 850 milligrams of procaine to cause death by respiratory paralysis in an average time of thirty minutes. In the head up position it takes from 900 to 1000 milligrams to result in death in an average time of fifty minutes. In the head down position 400 milligrams was followed by death in an average time of seventeen minutes. He concludes that the Trendelenburg position is not only useless but harmful. It must be remembered that CoTui was not dealing with therapeutic doses. The concentrations were tremendous and the specific gravity must of necessity have been extremely high.

Labat's theory behind the use of the Trendelenburg position to prevent certain dangerous complications incidental to the fall in blood pressure accompanying the anesthesia was to prevent cerebral anemia. His feeling was that with the tremendous vasodepression that occurs the one structure to suffer most was the brain. A fall in blood pressure, if severe, would allow the cerebral blood vessels to collapse, since they have weak walls unsupported by musculature. A collapse of these walls would be followed by cerebral anemia. To prevent this collapse he felt that to maintain these channels patent would be to forestall any such disastrous occurrence and the best way to maintain patency most dependably was to take advantage of the increased blood flow to the brain that the Trendelenburg position would give. CoTui, in reaching his conclusion that the Trendelenburg position was both useless and harmful performed no experiments to learn if there was any basis of fact in Labat's theory.

Sise[41] feels that in this position the blood gravitates toward the vital nerve centers and hydrostatic action tends to maintain a higher pressure in them than is present in other parts of the body. He[40] agrees with many others that operations should always be done in this position as far as possible, except when using a heavy solution. He[41] states as to the disadvantages of this position that if there are large masses of abdominal fat, the movements of the diaphragm may be impeded and respiration hampered. Woodbridge[47] institutes this procedure in the presence of a falling pressure. Stout[42] employs it as demanded to prevent cerebral anemia.

Tables 3, 4 and 5 represent summation of techniques by several workers with different agents. In the procaine series, table 3, we see that Labat employs 150 milligrams as a maximum dose, uses no ephedrine, varies the volume of the injected fluid but slightly, employs Trendelenburg position immediately, and varies the site of puncture. Evans, using 120 milligrams as maximum, also employs no ephedrine, substituting adrenalin, and applies the Trendelenburg position, if vasomotor collapse occurs or if the anesthesia is too high. He varies the volume injected much more than does Labat and keeps his punctures lower. Stout introduces more procaine into the spinal canal, administers ephedrine in varying doses, depending on the height of anesthesia, keeps the injected volume low, institutes Trendelenburg position if necessary and utilizes the same interspace for all levels of surgery. He modifies the duration of the injection depending on the level of anesthesia desired. Vehrs employs greater doses than any of the others here, administers no ephedrine, varies the dilution slightly, employs varying degrees of the Trendelenburg position and sites of puncture depending on height of anesthesia desired. Rovenstine's technique for transurethral surgery consists of moderate dosage plus dextrose solution, no ephedrine, reverse Trendelenburg position and low puncture.

Thus Labat depends on varying levels of puncture, changes in dosage and the Trendelenburg position for height of anesthesia; Evans, on varying dosage, volume and site of puncture; Stout, on varying dosage and speed of injection; Vehrs, on varying dosage, volume and site of puncture.

In the nupercaine series, Table 4, we see that Keyes, using the 1:200 solution, employs an unvarying dose, site of puncture and position but varies the volume injected. His dosage is full, for he[15] states that the maximum therapeutic dose may not be far from 2 cc. of a 1:200 solution. On occasion he recommends 50 milligram doses of ephedrine. Jones, administering the 1:1500 solution, varies dosage, volume and site of injection, as does Ehrlich. Wilson varies dosage and volume but not the site of injection. He injects his patients in the sitting position and varies the duration of this posture. Romberger blocks all cases for abdominal surgery to the nipple line with this nupercaine-procaine mixture, varying dosage, volume slightly, position of patient and site of puncture. Recently he has increased his nupercaine dosage slightly[37].

Table 5, Woodbridge, with metycaine, varies dose, volume and position of the patient. Meeker and McCreary[29] with this drug use 100 milligrams for lower and midabdominal operations. They give no dosage for upper abdominal operations, utilize barbotage but do not state total dilution.

McCuskey, with 1 per cent pantocain in saline, varies dosage, volume and site of puncture. He gives as dosage for pantocain[30] 1 milligram for each ten pounds of body weight plus 5 milligrams. The technique consists of diluting the 1 per cent solution with an equal quantity of spinal fluid so that the concentration as it enters the dural canal is 0.5 per cent. Since this

does not make much volume he advises higher punctures than when using procaine.

INDICATIONS

In considering the indications of a method one of the best arguments against its use is unfamiliarity with either the agent or with the technique involved. When faced with choosing between two methods or agents the anesthetist is and should often be guided by this and administer the agent or procedure in which he has the greater confidence. An expertly administered ether anesthesia in the presence of a pulmonary condition may often be less disastrous than a spinal anesthesia poorly administered.

Operative Site.

To list a column of indications for this method is not particularly satisfactory since so many factors enter into play. One can but generalize.

1. Operations on the lower extremity often indicate this method. Operations on the lower leg may be done under regional anesthesia, if the anesthetist is skilled in this latter method.

2. Operations on the perineum, rectum and external genitalia may be safely performed under either spinal or sacral anesthesia, depending again on the anesthetist.

3. All intra-abdominal surgery may also indicate the procedure.

4. Operations on the head, neck and thorax. Spinal anesthesia was suggested as possible for surgery in these areas by Koster and Kasman[17]. Their feeling was based on experiments carried out by them where neocaine was applied to the cervical cord and medulla of animals resulting in general anesthesia with no interference with respiration. CoTui[5], however, was able to demonstrate that respiratory stoppage due to paralysis of the respiratory center does occur when procaine is introduced into the subarachnoid space in sufficient dosage. It is generally conceded that attempts to produce as high an anesthesia as is necessary for surgery of the thorax and higher are dangerous.

Condition of Patient.

Lundy[25] feels that spinal anesthesia is contraindicated in all debilitated patients. Yet it does seem as though poor risk patients for perineal or lower extremity surgery do better than they would under other types of anesthesia.

Patients with advanced cardiac or vascular disease do not seem to do well, nor do patients with extremes in blood pressure. Where there exists a low functioning kidney condition and relaxation is necessary, spinal anesthesia often saves the added strain on this organ that might occur as result of ether anesthesia. Spinal anesthesia in place of ether in diabetics is very satisfactory.

The stout strong patient is often a better subject under spinal anesthesia than he would be under an inhalation method. The extremely apprehensive patient, unless heavily medicated, is usually a poor subject. CoTui[2] in animal experiments showed that, "pathologic states as dog pneumonia, postoperative infections and low blood pressure states . . . produce a marked reduction in the lethal dose". He pointed out the possibility that anoxemia might be a responsible factor in reduction of the lethal dose.

Because of the relaxation produced by the method one is tempted to employ it under certain conditions. In intestinal obstruction spinal anesthesia is a great aid to the surgeon allowing better exposure and closure. In advanced intestinal obstruction where the operative field is to be limited as for ileostomy a simple field block would be the better choice of anesthesia. The surgeon must be familiar with the increased intestinal activity produced by spinal anesthesia and allow for intestinal drainage, where possible. Some workers feel that mechanical obstruction is a contraindication. Upper abdominal surgery, particularly in the healthy adult, is facilitated by this method. Because of the hyperperistalsis occasioned by spinal anesthesia it has been recommended in the treatment of paralytic ileus. Here splanchnic anesthesia, administered by the posterior route described by Kappis, is to be preferred[34].

Patients suffering from shock or hemorrhage do not usually fare well under this procedure.

Indications for the Drug.

Procaine should be the drug most often used under all ordinary conditions. The forthcoming article on metycaine by Woodbridge may result in replacing procaine, because of its slightly longer action.

When it is necessary to employ an agent to give a more lasting anesthesia than either of the above it is necessary to turn to either pantocain or nupercaine. It must be remembered that because of the greater action of pantocain on the respiratory mechanism its dose should be kept down as low as possible and that its use should be restricted to those cases where a safe dose of procaine will not produce anesthesia of sufficient duration[30]. Essex and Lundy feel that pantocain in a selected group of cases is the anesthetic of choice for long operations in the lower abdomen.

Nupercaine causes a lengthened period of induction of anesthesia. The addition of procaine, by method of Romberger, would shorten this. Nupercaine is claimed by some to have a greater tendency to cause postoperative headache than are the other agents.

In patients in whom it is desired to disturb the blood pressure as little as possible either pantocain or nupercaine may be indicated.

SUMMARY

A review of the agents and methods of and indications for spinal anesthesia is offered with an effort to contrast their several characteristics and an attempt to present the various views on some disputed aspects.

REFERENCES

1. Babcock, W. W.: Fact and fancy in relation to spinal anesthesia. Tr. Meet. Am. Soc. Regist. Anesth. **1:** 4 (April) 1933.
2. CoTui, F. W.: Effect of pathologic states on minimum lethal dose of procaine intracisternally. J. Pharmacol. & Exper. Therap. **50:** 51 (Jan.) 1934.
3. Ibid.: Effect of different narcotics and narcotic combinations on minimum lethal dose of procaine hydrochloride intracisternally. J. Pharmacol. & Exper. Therap. **48:** 229 (June) 1933.
4. Ibid.: Further studies in subarachnoid anesthesia. Anesth. & Analg. **13:** 143 (July-Aug.) 1934.
5. Ibid.: Experimental studies on subarachnoid anaesthesia; effect of procaine hydrochloride on respiratory center. New York State J. Med. **33:** 391 (March 15) 1933.
6. Ibid.: Further studies in subarachnoid anesthesia. Anesth. & Analg. **13:** 183 (Sept.-Oct.) 1934.
7. Davis, L.; Haven, H.; Givens, J. H., and Emmet, J.: Effects of spinal anesthetics on spinal cord and its membranes; experimental study. J. A. M. A. **97:** 1781 (Dec. 12) 1931.
8. Ehrlich, S. D.: Spinal anesthesia with "hypobaric" solution of nupercain. Anesth. & Analg. **13:** 240 (Nov.-Dec.) 1934.
9. Essex, H. E., and Lundy, J. S.: Experiments with anesthetics: I. A method of testing local anesthetics by cisternal injection of frogs. Proc. Staff Meet. Mayo Clin. **6:** 227 (April 15) 1931.
10. Israëls, M. C. G., and MacDonald, A. D.: The pharmacology of percaine. Brit. M. J. **2:** 986 (Nov. 28) 1931.
11. Jarman, R.: Experiences with spinal anesthesia. Tr. Am. Soc. Regist. Anesth. (Oct. 9) 1934.
12. Jones, W. H.: Percain; new regional and spinal analgesic, with special reference to high thoracic nerve root block and new technique. Anesth. & Analg. **9:** 218 (Sept.-Oct.) 1930.
13. Ibid.: Subarachnoid block general analgesia: "spinal" anesthesia: respiratory paralysis fallacies and methods. Anesth. & Analg. **11:** 224 (Sept.-Oct.) 1932.
14. Kemp, W. N.: A résumé of spinal anesthesia. Am. J. Surg. **19:** 534 (March) 1933.
15. Keyes, E. L., and McLellan, A. M.: Further experiences with nupercaine. J. A. M. A. **96:** 2085 (June 20) 1931.
16. Kolmer, J. A.: Approved Laboratory Technique. New York: D. Appleton Company. 1931. p. 236.
17. Koster, H., and Kasman, L. P.: Spinal anaesthesia for the head, neck, and thorax; its relation to respiratory paralysis. Surg. Gynec. Obst. **49:** 617 (Nov.) 1929.
18. Ibid.: Histologic studies of spinal cord following spinal anesthesia. Am. J. Surg. **25:** 277 (Aug.) 1934.
19. Labat, G.: Regional Anesthesia: Its Technic and Clinical Application. Philadelphia: W. B. Saunders Company, 1924.
20. Lindemulder, F. G.: Spinal anesthesia: its effect on the central nervous system. J. A. M. A. **99:** 210 (July 16) 1932.
21. Lundy, J. S.; Essex, H. E., and Kernohan, J. W.: Experiments with anesthetics. IV. Lesions produced in the spinal cord of dogs by a dose of procaine hydrochloride sufficient to cause permanent and fatal paralysis. J. A. M. A. **101:** 1546 (Nov. 11) 1933.
22. Ibid.: Advances in anesthesia from the standpoint of the anesthetist. J. A. M. A. **99:** 968 (Sept. 17) 1932.
23. Ibid.: Outline of some of the advantages and disadvantages of various agents used in anesthesia. Minnesota Med. **15:** 219 (April) 1932.
24. Lundy, J. S.; Essex, H. E., and Kernohan, J. W.: Experiments with anesthetics; lesions of spinal cord in dogs produced by dose of procaine sufficient to cause permanent and fatal paralysis. Proc. Staff Meet. Mayo Clin. **8:** 88 (Feb. 8) 1933.
25. Lundy, J. S.: General, local, spinal and balanced anesthesia. Minnesota Med. **16:** 572 (Sept.) 1933.
26. Marvin, F. W.: Pantocain in spinal anesthesia. New Eng. J. Med. **207:** 368 (Aug. 25) 1932.
27. Ibid.: Present status of various spinal anesthetics and their clinical usefulness. J. A. M. A. **101:** 1475 (Nov. 4) 1933.
28. Maxson, L. H.: Spinal anesthesia: technique, records and results. Anesth. & Analg. **13:** 26 (Jan.-Feb.) 1934.
29. Meeker, W. R., and McCreary, P. L.: Metycaine in spinal anesthesia. Am. J. Surg. **24:** 139 (April) 1934.
30. McCuskey, C. F.: Pantocain as spinal anesthetic. Anesth. & Analg. **12:** 116 (May-June) 1933.
31. Middleton, W. S., and Chen, K. K.: Ephedrine: a clinical study. Arch. Int. Med. **39:** 385 (March) 1927.
32. Nonne, M., and Demme, H.: Degenerative Myelitis nach Spinal-Anästhesie. Wien. klin. Wchnschr. **41:** 1002 (July 12) 1928.
33. Nowak, S.: Personal Communication.
34. Ochsner, A., and Cutting, R. A.: The treatment of ileus with splanchnic and spinal anesthesia. Anesth. & Analg. **9:** 91 (March-April) 1930.
35. Phelps, A. E.: Evaluation of cardiac and circulatory stimulants for surgical patients. Ann. Surg. **91:** 24 (Jan.) 1930.
36. Romberger, F. T.: Combined novocaine-nupercaine spinal anesthesia. Am. J. Surg. **11:** 471 (March) 1931.
37. Ibid.: Personal Communication.
38. Rovenstine, E. A.: Anesthesia for transurethral prostatic resection; comparative study of transsacral and spinal blocks. J. Urol. **31:** 633 (May) 1934.
39. Rudolf, R. D., and Graham, J. D.: Notes on sulphates of ephedrin. Am. J. M. Sc. **173:** 399 (March) 1927.
40. Sise, L. F.: Spinal anesthesia fatalities and their prevention. New Eng. J. Med. **200:** 1074 (May 23) 1929.
41. Ibid.: Vascular depression of spinal anesthesia. S. Clin. of North America. **9:** 1369 (Dec.) 1929.
42. Stout, R. B.: Spinal anesthesia: volume control technique. Am. J. Surg. **7:** 57 (July) 1929.
43. Thomson, H. T.: Spinal anesthesia. Edinburgh M. J. (April) 1931.
44. Vehrs, G. R.: Spinal Anesthesia: Technic and Clinical Application. St. Louis: C. V. Mosby Company. 1934.
45. Wiedhopf, O.: Pantokain, ein neues Lokalanästhetikum. Deutsche Med. Wchnschr. **57:** 13 (Jan. 2) 1931.
46. Etherington-Wilson, W.: Intra-thecal nerve root block: some contributions and a new technique. Brit. J. Anesth. **11:** 43 (Jan.) 1934.
47. Woodbridge, P. D.: Neothesin for spinal and regional anesthesia: preliminary clinical report. Anesth. & Analg. **11:** 177 (July-Aug.) 1932.
48. Ibid.: Personal Communication.

A COMPARISON OF POSTOPERATIVE COMPLICATIONS FOLLOWING GENERAL AND SPINAL ANESTHESIA

BY BORIS RAPOPORT, M.D.*

IN spite of recent advances in anesthesia, postoperative pulmonary complications attributable to the anesthetic agent still form a perplexing problem. For a time it was believed that spinal anesthesia would solve this troublesome condition, because "ether pneumonia", "inhalation pneumonia" and pulmonary atelectasis due to mucus plugs in any part of the bronchial tree were entirely associated with the administration of general anesthesia. Experience, however, has compelled us to change our views in this matter. We have learned that there are many other factors, besides the anesthesia, which are conducive to postoperative complications. Statistics have proved beyond doubt that spinal and local anesthesia do not render the patient immune against such complications.

King[1] reported a series of cases from the surgical service of the Massachusetts General Hospital, and came to the conclusion that statistically the type of anesthesia bore no relation to the development of complications. McKittrick, McClure, and Sweet[2], who published a review at some previous date from the same hospital, of postoperative pulmonary complications following spinal anesthesia as compared with general, proved that the percentage of complications increased with spinal anesthesia. On

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the other hand, Brunn and Brill[3] reported a number of cases where the pulmonary complications after operation were three and a half per cent larger under general anesthesia than under spinal. Judging from these varied reports it would seem that no two clinics agree as to percentage of complications under various anesthetics.

The practice of spinal anesthesia since its inception about thirty-five years ago has been marked by cycles of undue enthusiasm mixed with periods of criticism and censure. It is a well-known fact, however, that a well-developed method and a familiar drug in the hands of a few cannot always prove successful in the hands of the many. We must also take into consideration that the reported statistics do not take into account the condition of the patients before operation and the number of cases which had to be supplemented with a general anesthetic. A certain small percentage of deaths from anesthesia is unavoidable. It cannot be denied, however, that spinal anesthesia has in most cases been beneficial to all concerned: surgeon, anesthetist and patient. Also that many difficulties in patients with pulmonary conditions, nephritis, diabetes, and in some cases even cardiac diseases were easily solved by the administration of spinal anesthesia.

There is practically no contraindication to spinal anesthesia. More than half of all operations below the diaphragm can be performed with as low as 50 to 100 mgm. of novocain, which is considered a safe dose. Formerly it was believed that patients with a low blood pressure should not be operated upon under spinal anesthesia. Now the tendency is to consider hypertension as a contraindication. It seems, however, from actual experience that neither condition should rule out this valuable method of anesthesia, since in numerous cases spinal anesthesia has been administered by me to patients with systolic pressures as low as 80 and as high as 250 without any bad effects.

As a rule, given a perfectly well patient having to undergo an operation of moderate severity, spinal anesthesia will cause very few complications. It is only in the sick patient or the severe operation that spinal anesthesia contributes its share of complications. But that share is much smaller than under general anesthesia, as will be shown in the following tables.

COMPLICATIONS FOLLOWING GENERAL ANESTHESIA

	No. of Cases	Complications No.	Per Cent	(Pulmonary Comp.) No.	Per Cent	Deaths No.	Per Cent
Gall Bladder operations	42	8	(19%)	6	(14.3%)	3	(7.1%)
Hysterectomy	95	11	(11.5%)	7	(7.3%)	1	(1%)
Herniorrhaphy	50	5	(10%)	2	(4%)	2	(4%)
Kidney operations	14	1	(7%)	0		0	
Appendectomy	94	9	(9.5%)	7	(7.6%)	4	(4.2%)
Total	295	34	(11.5%)	22	(7.4%)	10	(3.4%)

COMPLICATIONS FOLLOWING SPINAL ANESTHESIA

	No. of Cases	Complications No.	Per Cent	(Pulmonary Comp.) No.	Per Cent	Deaths No.	Per Cent
Gall Bladder operations	104	13	(12.5%)	8	(7.7%)	5	(4.4%)
Hysterectomy	50	2	(4%)	0		0	
Herniorrhaphy	83	7	(8.4%)	6	(7.2%)	2	(2.1%)
Kidney operations	80	4	(5%)	4	(5%)	3	(3.7%)
Appendectomy	13	1	(7.6%)	1	(7.6%)	0	
Total	330	27	(6.5%)	19	(5.7%)	10	(3%)

These tables comprise the records of five types of major operations performed at the Beth Israel Hospital, Boston during the years 1931 and 1932. The operations selected are the following: appendectomies, hysterectomies, herniorrhaphies, cholecystectomies and kidney operations. In the latter group the year 1933 was also included. These operations were chosen as both types of anesthesia were in nearly equal numbers represented in the total series. Patients under fifteen years of age were excluded, since spinal anesthesia was not administered to children. A total of 625 cases were studied consecutively: 238 males and 387 females; general anesthesia 295, spinal 330.

By comparing the two tables it can readily be seen that patients operated upon under spinal anesthesia fared much better than those under general. While the complications under general anesthesia were 11.5 per cent (pulmonary 7.4 per cent), those of spinal anesthesia comprised only 6.6 per cent (pulmonary 5.7 per cent). The percentage of deaths under general was 3.4 per cent, while under spinal it was 3 per cent. At the same time we must also take

into consideration the fact that, while the percentage of medical ailments before operation in patients operated under general anesthesia was only 7 per cent, those done under spinal amounted to 25.5 per cent.

There is no dispute, even among those opposed to spinal anesthesia, that for relaxation and absence of aftereffects there is no equal to it. Genito-urinary and orthopedic operations lend themselves more conveniently to spinal than does abdominal surgery. Lower abdominal surgical procedures are much more successfully accomplished than upper abdominal. The chances of successful anesthesia in surgery of the chest, neck and head, is greatly reduced, in spite of the number of cases reported; while the element of danger is markedly increased. It is therefore wise to limit spinal anesthesia to operations below the diaphragm.

The only drawbacks of spinal anesthesia are the limited time to which the surgeon is confined for the completion of the operation and the nausea and vomiting which frequently hamper its progress. Both of these difficulties, however, can be materially aided by appropriate preliminary medication. Sodium amytal and nembutal in balanced doses have a tendency to suppress the vomiting reflex and to diminish the patient's sensitivity to pain. These drugs, which are given by mouth about an hour and a half before operation, may be augmented by morphin hypodermically half an hour before. The employment of a solution of pantocain has in many cases given a much longer and deeper anesthesia than the crystals. It seems, however, that it is slightly more dangerous than the latter, since it has a greater tendency to paralyze the muscles of respiration.

The question of supplementary anesthesia after the spinal anesthesia wears off has been troublesome. Where no relaxation is required gas-oxygen serves as a good adjunct. In abdominal operations, however, no relaxation can be obtained from the addition of gas-oxygen, and the greatest difficulty is met with during the closure of the peritoneum. In these cases injection of novocain into the abdominal muscles at the sides of the wound by the surgeon facilitates greatly the suturing of the abdominal wall. This can also be augmented by slow drop ether on an open mask, sufficient to daze the patient, but not pushing the ether to the point of complete relaxation.

We must not, however, overlook the fact that sudden fatalities, chiefly soon after the injection of the anesthetic solution, do occur. Some of these seem to be caused by respiratory paralysis and others may be due to cardiac failure. The theory of toxic absorption of novocain, described by Nowak[4] in his experiments, seems reasonable only in cases where after fifteen or twenty minutes a severe reaction sets in, manifested by a marked drop in blood pressure, slowing of pulse, nausea, vomiting, restlessness, and profuse perspiration with pallor. This, however, does not explain the immediate reactions manifested by respiratory or cardiac embarrassment, which must be due either to direct action of the drug on the respiratory centre or to the innervation of the heart.

Since my last report (in June, 1931[5]) of 1875 operations performed at the Beth Israel Hospital under spinal anesthesia we have added 1402 more operations under the same anesthetic. In this last series we had two deaths in which the spinal anesthesia was to all appearances the sole causative factor.

CASE 1. A fifty-six year old female patient with myocarditis, slightly enlarged heart, and blood pressure of 180/100, was to undergo an operation on the knee joint. The injection was done in the third lumbar interspace with the patient in the sitting position, using an ephedrin-novocain solution as a local anesthetic. One hundred and twenty-five mg. of novocain crystals were employed for the spinal anesthesia. The patient was put on her back in slight Trendelenburg position. About two minutes after the injection of the solution the patient began to complain of a "weakness of the heart". She soon became pulseless, but still kept on breathing slowly for about one minute and died. Heart stimulants and artificial respiration were of no avail.

CASE 2. A male patient, aged sixty-seven. Physical examination revealed hypertension with weak heart sounds, râles at the left base with a question of old tuberculosis, and a slight trace of albumin with casts in the urine. He was to undergo a second-stage prostatectomy, the first stage having been done under spinal anesthesia about ten days previously. The injection was done in the fourth lumbar interspace with the patient in the sitting position. As in the first stage 75 mg. of novocain crystals were used. The patient began to complain of difficulty in breathing soon after he was placed on his back. The anesthesia did not extend above the umbilicus, his blood pressure remained practically the same, 165/90, and his pulse rose from 84 to 108. There was no cyanosis. In view of the patient's good general condition it was decided to go on with the operation, which lasted about fifteen minutes. Soon after the operation the patient became cold and clammy and his finger tips were cyanosed. His pulse and blood pressure did not as yet undergo any material change. He was put to bed and given 500 cc. of ten per cent glucose in saline intravenously. His respiration became more embarrassed, cyanosis increased, and his pulse began to show slight weakness and irregularity. The blood pressure dropped to 115/75. Stimulants were administered and for a while he rallied. However, he soon became more cyanosed, gasping respiration developed, and he lapsed into unconsciousness and died. Death occurred about one and one-half hours after the spinal anesthesia was induced.

In the first case it was apparent that the cardiac factor was the more pronounced in the fatal termination, while in the second case respiratory embarrassment was more manifest as a cause of death. Since these two catastrophes the sitting position for the administration of spinal anesthesia has been abandoned and in the last 750 cases we have had no fatality.

So far the ideal drug, which should give a lasting and safe spinal anesthesia, has not been discovered. But, with proper technique, careful selection of the patient, gradation of the dose of the least dangerous drug, and the right care during and after operation, the mortality from this procedure should be reduced to a minimum and the complications brought down much below those of general anesthesia.

SUMMARY

1. The postoperative complications following spinal anesthesia are greatly exaggerated, when compared with those of general anesthesia.
2. In certain cases, where a general anesthetic would present some difficulties or danger, spinal anesthesia offers the best solution.
3. Effective preliminary medication suppresses vomiting and allays the patient's apprehension. It also aids in prolonging the anesthesia.
4. Should the anesthesia wear off before the operation is completed, then gax-oxygen serves as a good supplement in cases where no relaxation is required. In abdominal operations, the injection of novocain into the muscles at the sides of the wound facilitates the closure of the peritoneum.

REFERENCES

1. King, D. S.: Postoperative pulmonary complications. II. Carbon dioxide as a preventive in a controlled series. J. A. M. A. **100:** 21 (Jan. 7) 1933.
2. McKittrick, L. S.; McClure, W. L., and Sweet, R. H.: Spinal anaesthesia in abdominal surgery. Surg. Gynec. & Obst. **52:** 898 (April) 1931.
3. Brunn, H., and Brill, S.: Observations on post-operative pulmonary atelectasis. Ann. Surg. **92:** 801 (Nov.) 1930.
4. Nowak, S. J. G.: Urinary excretion of novocain after spinal anesthesia and theory of toxic absorption. Anesth. & Analg. **12:** 232 (Nov.-Dec.) 1933.
5. Rapoport, B.: Observations on spinal anesthesia, with report of 1875 cases. New Eng. J. Med. **204:** 1254 (June 11) 1931.

MEDICAL PROGRESS

PROGRESS IN NEUROLOGY IN 1934

BY JULIUS LOMAN, M.D.*

ONE of the most important contributions to the neurological literature of 1934 is Brodie and Elvidge's[1] experimental work on poliomyelitis establishing the portal of entry and neurogenic distribution of the virus. Petersen's[2] studies on the effect of weather in nervous and mental diseases are intriguing and may open a new field for interpreting many of the symptoms of such diseases as migraine, multiple sclerosis, and tabes. Interesting physiological studies on epilepsy, migraine, and studies on the chemistry and treatment of the muscular dystrophies continue. The results of the intensive investigations on the 1933 St. Louis epidemic of encephalitis with important data of etiology, epidemiology, and pathology are reported. In addition, the neurological literature of 1934 contains many interesting descriptions of clinical conditions and a few new therapeutic procedures.

EPILEPSY

Experimental Studies

Gibbs, Lennox and Gibbs[3] have investigated the possible etiological rôle of vascular spasm with consequent cerebral anemia in epilepsy. By means of a thermoelectric blood flow recorder, inserted through a hollow needle into the internal jugular vein of epileptic patients, the cerebral blood flow could be observed before and during seizures. In none of the ten patients studied was there a significant reduction in blood flow immediately preceding the attacks. During several convulsions the blood flow increased markedly. It was concluded that this change is the result of, rather than a cause of, the seizures. These experiments strongly indicate that vascular spasm is not the important precipitating factor in cases of epilepsy.

Experiments by Finesinger and Cobb[4] tend to corroborate the above authors' results. Using the skull window technique of Forbes and measuring the diameter of the pial vessels following the administration of convulsants, Finesinger and Cobb observed in cats that large intravenous injections of caffeine caused marked convulsions which were preceded by vasoconstriction, a fall in systemic blood pressure and a decrease in cerebrospinal fluid pressure. Similar changes were noted following intravenous injections of absinth and homocamphin. On the other hand, monobromated camphor convulsions were preceded by dilatation of the pial vessels. The authors conclude that since fits caused by drugs are produced in the presence of either vasoconstriction or vasodilatation, the state of the blood vessels is not the essential factor. The authors believe that the drugs probably cause the convulsions by direct action on the nerve cells in some way as yet unknown.

In studies on mineral metabolism in young epileptics, Engel, McQuarrie and Ziegler[5] point out the dependence of epileptic attacks on water and mineral metabolism. To illustrate this dependence, two interesting cases are cited. In the first case, a child who was given pituitrin to create a positive water balance, the amount of urine excreted became diminished and in the pituitrin-free periods, the amount of urine excreted became greater than that of the fluid taken in. Furthermore, under the influence of

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pituitrin, the amount of chlorides excreted was considerably greater than the amount taken in. Sodium followed a similar but less striking course. Potassium, too, was increased in the urine under the influence of pituitrin. Usually calcium and magnesium were uninfluenced. Such disturbances in water and salt balance following pituitrin injections occur both in normal and in epileptic patients, but only in the latter are attacks produced. The above authors also showed that when the mineral loss is balanced by the addition of sodium chloride, a much greater retention of water with much more pituitrin is required to bring on an attack. On the other hand, if the diet is restricted as to sodium chloride, the pituitrin experiment acts much more quickly in producing an attack. In the other case, the diet was kept as low as possible in sodium chloride and potassium. After three days no more attacks occurred. Then when sodium chloride was added in large amounts, the body weight rose and one severe attack occurred on the same day. Here the retention of salt water precipitated the attacks. In the first case cited, the great loss of minerals with the simultaneous retention of water precipitated the attacks.

Since it has been demonstrated that retention of fluid in the body tends to induce seizures, Jacobsen[6] has used the following test to confirm or exclude the diagnosis of epilepsy: To produce a positive water balance Jacobsen gives 300 cc. of water every two hours and an injection of pitressin (containing the antidiuretic principle of pituitrin without smooth muscle stimulating principle) every four hours—0.2, 0.3, 0.4 cc., and thereafter 0.5 cc. until a seizure occurs or until eight to ten doses have been given. Jacobsen has found the test reliable in a series of forty cases. If the condition is epilepsy, a typical seizure will occur when sufficient fluid has been retained to raise the body weight by three to six per cent.

MIGRAINE

Physiological Studies

In his studies on the "patient and the weather", Petersen[2] associates migraine (and headache in general) with a sudden alkalosis (increased Ph with a reduction of CO_2 content of the blood) with vascular spasm, and in some cases with a "potassium leakage of the blood". He points out that the migraine equivalent is also associated with similar chemical and spastic states. Petersen also found that the onset of the attacks is meteorologically conditioned, occurring in most instances with great regularity, with polar infalls (inflow of cold polar air which occurs in frequent intervals in the region of the American storm tracts). In some instances, the headache occurs with a barometric down-turn from a high stable barometric pressure or sometimes at the first up-turn of the barometric pressure from a barometric low.

Relationship between Migraine, Epilepsy and Some Other Neuropsychiatric Disorders

Paskind[7] points out very strongly, after a review of the literature, that the studies of those who have stressed an epilepsy migraine relationship are largely vitiated by the lack of controls. This author compared the incidence of migraine among patients with epilepsy, among patients without neuropsychiatric disorders and those with manic-depressive psychosis, trigeminal neuralgia, psychasthenia, dementia praecox, tic, constitutional inferiority and paranoid states. The records of 3,326 patients were studied. The findings were as follows: In the group with non-neurologic disorders, migraine was found in the family in 14.4 per cent of cases; in the parent in 10.2 per cent; and in the patient in 3.3 per cent. In the group of epileptic patients, the percentage of migraine was much higher, being found in the family in 35.2 per cent; in the parent in 30.8 per cent; and in the patient in 8.4 per cent. There was, however, even a greater association between migraine and trigeminal neuralgia, being found in 37.7 per cent, 30.1 per cent and 20.3 per cent in the family, parent, and patient respectively. Again, there was a greater incidence of parental migraine in tic and in constitutional inferiority than in epilepsy. Thus, it is concluded that there is no especial relationship between migraine and epilepsy; that migraine occurs as evidence of a familial neuropathic trend in other neurologic conditions.

Treatment of Migraine

Lennox[8] studied the effect of ergotamine tartrate (gynergen) on thirty-nine men and six women. In forty of the cases abrupt termination of the headache occurred; in three there was no relief, and in two an apparent shortening of the attack occurred. In most instances, before the headache began to be lessened, nausea and vomiting, or both, occurred. When the drug was given intravenously relief occurred in fifteen to thirty minutes; when given subcutaneously in one to two hours; and when given orally in one to three hours. Relief of severe headaches was rare when the drug was given orally. The mechanism by which ergotamine relieves migraine is unknown. Many authors accept the theory of vascular spasm and assume that the drug paralyzes the motor sympathetic fibers, thereby relieving the spasm and pain.

The experiments of Lennox, Gibbs and Gibbs[9] throw light on the above theory. By means of a thermo-electric blood flow recorder, inserted into the internal jugular vein, injections of ergotamine tartrate in unanesthetized patients were observed to cause a moderate increase in cerebral blood flow which is readily explained by the concomitant increase in systemic blood pressure. Since ergotamine which relieves headache increases blood flow only moderately,

whereas adrenalin injections and carbon dioxide inhalations which do not uniformly relieve headache increase cerebral blood flow enormously, the authors conclude that the beneficial effect of the drug cannot principally be due to its action on cerebral blood flow.

Brock, O'Sullivan and Young[10], who tried the effect of various drugs on migraine, found the most striking benefit was obtained by ergotamine. Various drugs, such as mecholin, histamine (a very active cerebral vasodilator), amyl nitrite (another vasodilator), and adrenalin were unsuccessful in relieving the headache. The authors believe that vasospasm is probably a secondary effect of, and not a primary factor in, migraine.

Logan and Allen[11] obtained relief in sixty-seven out of seventy-one attacks of headache (nine individuals). Occasionally the reactions induced—nausea, numbness and pain in the extremities—were distressing. A trial subcutaneous injection of .25 mg. at the onset of the attack is recommended. If no untoward symptoms follow and the headache persists for two to three hours, a second injection of .5 mg. may be given without producing distressing reaction. The dose may be repeated every three to four hours to control the headache. Four to eight times the subcutaneous dose is required for oral administration.

VASCULAR DISTURBANCES

Cerebrospinal Hemorrhage in Purpura Hemorrhagica

Geiger[12] reports two cases of central nervous system involvement in this condition. In the first case, a twenty-four year old Italian woman, in addition to the usual symptoms and laboratory findings, severe persistent headache and a slight neck rigidity occurred. Three weeks from the onset of the headache, lumbar puncture showed thick, dark, red fluid. Amelioration of the symptoms followed. After four daily drainages, the spinal fluid cleared, and the symptoms steadily improved. Two months later symptoms recurred. Headache and vomiting was followed by paralysis of the left arm and leg. There were retinal hemorrhages, bleeding from the mouth and vagina, and purpuric spots. Platelets were absent. Before death, coma developed; all the limbs became spastic; the neck grew rigid; the right pupil became dilated and fixed; and the temperature rose to 104°. These symptoms suggested to the authors extensive hemorrhage within the cerebrum. In the second case, a twenty-four year old Irish woman presented the typical history and findings of purpura and developed coma and generalized convulsions. The coma deepened with Cheyne-Stokes respiration and death followed. These symptoms in the absence of localizing signs suggested either an intracerebral or large subdural hemorrhage.

Leukemic Changes in the Brain

In a histopathological study of fourteen cases of the various types of leukemia, Diamond[13] noted that the brain in every case showed definite changes; cerebral hemorrhage was found in three; in one man leukemic nodules were scattered through the brain; subdural hemorrhage was present in one case; petechial hemorrhage in two; and leukemic infiltrations (chloromas) were present in only one case. The meningeal and pial vessels were more or less infiltrated in every case. Leukemic infiltrations were observed in the cerebral vessels and capillaries. Degeneration of the ganglion cells with chromatolysis and neuronophagia were present in all cases. The glia also showed reactive phenomena. Necrosis, small cavities and softenings were not uncommon. Edema was present in eleven cases. The clinical symptoms were headache, noises in the head, dizziness, nausea, vomiting, ataxia, progressive weakness and generalized pains. In the acute cases there was restlessness, drowsiness, delirium and coma. In seven cases no definite neurologic or cerebral manifestations were in evidence.

Subdural Hemorrhage

Leary[14] points out that this condition is relatively common. Fifty brains were studied. In twenty-seven of the cases alcohol was a factor. When unaccompanied by skull fracture, the usual source of the hemorrhage is a ruptured bridging or subarachnoid vein. Alcohol is significant probably by causing more frequent traumas. The hemorrhage tends to be unilateral. Repeated hemorrhages may occur because a relatively avascular dura cannot organize a subdural clot.

In a paper on the diagnosis and treatment of subdural hematoma, Munro[15] gives the following salient features in sixty-two cases: Trauma is a chief cause. Other causes include diseases of the blood-forming structures, arteriosclerotic changes, syphilis, experimental avitaminosis, and nutritional changes in children. Diagnosis can be made only by exploration which is indicated in those cases in which adequate treatment for one of the commoner craniocerebral injuries fails to bring improvement. A history of head injury, followed almost always by unconsciousness, and often by headache, peculiar actions and speech are usually noted. A latent period with mild or no symptoms may vary in length up to years, when the clot is not too large or when the original injury is trivial. A little more than half the patients had a high intracranial pressure and a few, less than half, had evidence of subarachnoid bleeding. If improvement does not follow dehydration and lumbar puncture, the diagnosis should be confirmed or ruled out by a bilateral exploratory subtemporal trephine. The mortality in twenty-two proved cases, without surgery, was 100 per

cent and in thirty-nine cases, with operative treatment, was 41.2 per cent.

HEAD INJURY

In a paper on craniocerebral injuries, Lyerly[16] stresses the following rules in the management of these cases:

(1.) During the period of shock, the patient must be handled as little as possible and treatment should be limited to measures for overcoming the state of collapse.

(2.) After recovery from shock, a laceration of the scalp should be immediately débrided and sutured to prevent infection.

(3.) Pulse, respiration, blood pressure, condition of pupils, and state of consciousness should be watched to obtain early information regarding an increasing compression of the brain.

(4.) Depressed fractures of the skull should be operated on and the loose fragments of the bone replaced to close the cranial defect. They may not be replaced in a contaminated or even doubtfully infected wound.

(5.) Spinal drainage is indicated in cases with subarachnoid hemorrhage which gives signs of meningeal irritation.

(6.) A subtemporal decompression should be done when there are signs of localized pressure due to extradural or subdural hemorrhage. It should be done on the side of suspected pathology.

(7.) Dehydration may be used in all cases of brain injury; it is especially indicated when there is intracranial pressure due to trauma. It should, however, be used with caution. Too drastic dehydration is more harmful than a moderate edema.

In an analysis on the after-effects of head injury in 200 cases, in which 120 had postconcussional symptoms, Russell[17] points out that in the patients over forty years of age, the symptoms are apt to persist longer than in younger patients. Headache and dizziness are the most common symptoms. In patients over fifty, loss of memory and mental ability stand first. In children, nervousness and changes in behavior and personality are common effects. Epilepsy developed in 3.5 per cent.

ENCEPHALOGRAPHY

By the increasingly widespread use of this technique, a great deal of information is added to that gained by routine neurological examination. Not only does its usefulness lie in the diagnosis of cerebral neoplasms, degenerative diseases of the central nervous system, birth injuries and epilepsies, but serves in many cases to put on a true organic basis the symptoms following cerebral trauma which otherwise would be classified as a posttraumatic neurosis. Thus, the symptoms of headache, dizziness, personality changes, inability to concentrate are not infrequently explained by the characteristic encephalographic changes, such as arachnoiditis, with secondary atrophy and compensating hydrocephalus. Spinal insufflation of air may relieve the distressing posttraumatic symptoms. From a medicolegal point of view, the ability to demonstrate by the encephalogram the cerebral pathology has its importance in view of the usual lack of impressiveness with which the diagnosis of posttraumatic neurosis is received in court.

In the presence of increased intracranial pressure, posterior fossa neoplasms or in obstructive internal hydrocephalus, encephalography is contraindicated. In these conditions ventriculography is the method of choice. Encephalography in greatly debilitated individuals, in cases of severe myocarditis, hypertension and advanced arteriosclerosis carries risk.

Dyke and Davidoff[18] employ the following improved encephalographic technique: Lumbar puncture is done in the sitting position with the head of the patient resting against an upright Potter-Bucky diaphragm. Ten cc. of spinal fluid is withdrawn and five cc. of air is slowly injected. Alternate withdrawals of fluid and injections of air are carried out until twenty cc. of air has been insufflated, when a posterior-anterior film is taken. From this film may be determined the size, shape and position of the lateral and often the third ventricle, and the total amount of air necessary may be calculated. The above authors have found that fifty to seventy cc. of air is sufficient to outline the ventricles and subarachnoid space when the ventricles appear normal in the twenty cc. film. Anteroposterior, postero-anterior and right and left lateral stereoscopic films are done. By stereoscopic examinations not only the size, shape and position of all the cisterns but also their relation to neighboring structures may be ascertained.

INTRACRANIAL HYDRODYNAMICS

In an excellent paper based, in large part, on personal clinical investigations, Masserman[19] discusses the cerebral disturbances following rapid fluid decompression of the ventriculo-subarachnoid spaces. He stresses the rôle of cerebral edema in the production of distressing and sometimes serious symptoms following lumbar puncture. To test this hypothesis, Masserman gave to sixty-six patients, suffering from probable cerebral edema produced by lumbar tap, 250 cc. of twenty-five per cent intravenous glucose. Sixty-four obtained relief; sixteen slight or no relief; and six suffered an accentuation of their symptoms. In the control group receiving saline (for psychic effect), only eight out of forty-six attributed any therapeutic effect to the injection. Eight became worse and thirty became unaffected. These experiments demon-

strate the advisability of slow removal and careful pressure for pressure replacement of air in encephalography and ventriculography.

ENCEPHALITIS

Etiology, Epidemiology and Pathology

Further studies on the St. Louis epidemic of encephalitis were reported in 1934. Muckenfuss, Armstrong and Webster[20] were able to isolate a number of strains of a virus that seem to be the etiological agent of the 1933 epidemic. The virus acted on monkeys and white mice and appeared to be distinct from other previously known viruses. It was found that the number of strains of similar characteristics isolated and the neutralization of the virus by serum of individuals already recovered from the encephalitis in this epidemic but not by the serum of patients recovered from other diseases, justified the conclusion that it is the etiological agent of the recent epidemic.

Webster and Fite[21], as a result of bacteriological studies, conclude that the active agent of epidemic encephalitis is a specific virus. Gay and Holden[22] present evidence tending to incriminate a neurotrophic strain of the virus of herpes simplex as the causative agent of epidemic encephalitis. They were able to isolate the herpetic virus from five cases of encephalitis.

Leake, Musson and Chope[23], studying the epidemiology of the 1933 St. Louis encephalitis epidemic, point out that individual susceptibility, in which age plays a part, appears to be more important than contagion. There were few multiple cases in the same family. Water, milk and the mosquito were eliminated as the means of transmission.

Pathologically, according to McCordock, Collier and Gray[24], the only constant macroscopic alteration in the central nervous system of twenty-eight cases was vascular congestion of varying degrees. Microscopically, intense congestion of the vessels down to the finest capillaries was present. Small arachnoid hemorrhages and occasionally extravasation of blood into the perivascular spaces and into the substance of the brain, or petechial hemorrhage in the brain tissue, were found. Both perivascular, diffuse, or focal collections of various types of inflammatory cells were found. Various degrees of pathology were seen in the nerve cells.

Treatment of Epidemic Encephalitis

Neal[25] gives her experience in the case of a formalized herpes virus. In controlled experiments of a year, much better results were obtained with this virus than with a vaccine prepared from normal rabbit serum. In twenty-eight cases treated with the vaccine the mortality was lower than in the control group. The least percentage of improvement was found after use of Rosenow's serum. The author warns against the use of malarial therapy as being dangerous and considers other fever methods of little value. Bulbocapnine was found too toxic.

Mutch[26] recommends calcium aspirin in all forms of chorea on the following basis: The drug supplies calcium (which has been considered to be deficient in the body in chorea); it produces a sedative effect on the brain and combats the causes for the rheumatic disorder. The neutral solution (1 Gm. dissolved in 5 cc. of water) may be given intravenously, subcutaneously, rectally, or orally. It is tasteless and is not irritating to the kidney or stomach. Mutch treated nineteen patients who were kept in bed. The average daily dose of the drug varied up to forty-five Gm. for a child of twelve. The average time taken to control the chorea was seventeen days with limits from seven to forty-six days respectively.

Fate of Encephalitics

In a follow-up investigation of forty encephalitics who had survived the acute stage, Sauter[27] found that six had died in the interim; six were not heard from; thirteen cases were affected with parkinsonism; seven were in a pseudo-neurasthenic state; four showed subsequent disease (2 bradyphrenia, 1 character alteration and 1 epilepsy). The pseudo-neurasthenics were able to work and showed only slight signs of encephalitis. In most of the cases the memory was more or less affected. The disease was stationary in one-half of the cases.

MULTIPLE SCLEROSIS

Etiology

An attractive theory of this disease has been proposed by Petersen[2] in his discussion on the patient and the weather. He regards multiple sclerosis as meteorologically induced, primarily due to vasomotor spasm in tissues in which anoxemia leads promptly to injury in unstable individuals living in the northern latitudes, particularly in the region of the storm tracts. This concept would explain the clinical symptomatology: its transient and variable nature, the exacerbations and remissions. It also explains why such agents as nicotine, lead, ergot, cold, chilling, and dampness which may accentuate spasm, have been incriminated as etiological factors in multiple sclerosis.

Therapy

Weinberg[28] reports his results with lecithin treatment in multiple sclerosis. This treatment is based on the theory advanced by Marburg (in 1906), who believed that multiple sclerosis might be related to a disturbance in the fat metabolism of the nervous tissue. Weinberg treated twelve patients who had been resistant to other procedures. The therapy consists of four intraspinous injections, in ten to twelve day intervals, of .2 cc. of lecithin mixed in four

cc. of warm normal saline solution diluted with an equal amount of the patient's spinal fluid. The author states that in nine of the twelve cases, there was greater or less improvement. Three of the severer cases showed particularly gratifying results. The method is harmless, although headache and a rise in temperature follow the injections.

By means of diathermy, radiotherapy, or the electric blanket, Neymann and Osborne[29] have treated twenty-five cases of multiple sclerosis. Treatments were applied biweekly. A temperature curve with a quick rise to 103.5°F. and a high plateau, ranging between 105° and 105.5°, was considered the optimum of the therapeutic requisition. The temperature was maintained by enveloping the patient in air-tight bags. In four mild cases, improvement occurred to a point where without the history the clinical diagnosis would be difficult. In eleven advanced cases only two attained a practically normal gait. The other nine showed improvement in their gait but retained spasticity. Four returned to work and seven lived at home in a semi-invalid state. Four of six bedridden cases regained the use of their upper extremities. Of the entire advanced group two were much improved and four lost some of their more troublesome symptoms. The cases were observed for a period of a few weeks to eighteen months following treatment. The authors conclude that it is too soon to evaluate the results of the treatment.

THE MENINGITIDES

Acute Lymphocytic Meningitis

This syndrome, which is believed by many authors to be related to either poliomyelitis or epidemic encephalitis, is discussed by Abramson[30]. Following a period of one to two weeks during which time symptoms of an upper respiratory infection may be present, there occurs a sudden onset of headache, vomiting, mild fever, and a varying degree of rigidity of the neck and a mild Kernig sign. In adults blurring of the discs is fairly common. The spinal fluid characteristically shows a marked lymphocytosis varying from a few hundred to thousands. The fluid is clear. The illness is relatively short and the prognosis is good.

A similar syndrome is described by Viets and Watts[31] who report four cases. The onset in these cases was acute with meningeal signs and with lymphocytosis of the spinal fluid. The sugar and chlorides were found to be always normal, which differentiates the disease and tuberculous meningitis. Rarely coma and convulsions occur which disappear after the first or second withdrawal of the spinal fluid.

Cases of "Fatal" Meningitis with Recovery

Reports of recovery from various types of meningitis are given by several authors. Felsen and Osofsky[32] cite a case of streptococcus (viridans) meningitis which followed a head injury with laceration of the scalp. The spinal fluid contained 5,000 cells, ninety-six per cent of which were polymorphonuclear leukocytes. The sugar content was 16.6 mg. per cent; total protein .43 per cent. Because smears of the fluid showed cocci and a gram-positive bacillus, suggestive of the anaerobic group, gas gangrene serum was given intraspinally. Further bacteriological studies revealed pure culture of streptococcus (viridans). The serum was continued since improvement occurred. Four doses of five cc. each were given. The patient had a rapid convalescence. Immunologic studies showed that the gas gangrene serum by increasing the degree of phagocytosis inhibited the growth of the strains of streptococcus (viridans) isolated from the patient's spinal fluid and from his throat.

A case of recovery from cerebrospinal fever by treatment with intravenous injections of urotropin is reported by Chopra[33]. After injections of antimeningococcal serum was used with insufficient effects, five cc. of urotropin (40 per cent strength) was injected intravenously for twelve days. Improvement seemed to follow each injection. The patient was cured after about six weeks.

A case of Type IV pneumococcus meningitis, which recovered following continuous cisternal drainage, frequent lumbar punctures, cisternolumbar irrigations, cisternal irrigations, and periodic cerebral dehydration with hypertonic dextrose, is reported by Bedell[34]. Approximately 200 cc. of spinal fluid was drained from the cisterna magna every twenty-four hours.

Reveno and McLaughlin[35] report a case of pneumococcus meningitis, Types I and II, which recovered after intravenous and intraspinal injections of 120,000 units of stock Felton's antipneumococcic serum. Recovery occurred after seven days of treatment.

Spontaneous recovery from tuberculous meningitis in a fourteen-year old girl was observed by Parry[36]. Only one lumbar puncture, which showed the characteristic changes of tuberculous meningitis, including the presence of tubercle bacilli, was done. The patient remained well in every way. Her case was followed for four years.

SPINAL CORD

Anterior Poliomyelitis

By means of the following unique experiments on the Macacus rhesus monkeys, Brodie and Elvidge[1] demonstrated the portal of entry and pathway of the poliomyelitis virus into the brain: Nasal installations of cord containing virus failed to infect monkeys in whom bilateral section and partial removal of the olfactory bulb and tract were carried out; whereas control monkeys were paralyzed after single

nasal installations of the virus. Furthermore, it was demonstrated that although the nasal mucosa is innervated by the V. and VII. cranial nerves, as well as by the olfactory nerve, only the last named nerve transmits the virus to the central nervous system, for no infection occurred when the olfactory tract was cut. These animals received nasal inoculations over a period of three weeks, during which time much must have dribbled into the gastrointestinal tract, yet no infection occurred. This observation would tend to eliminate the gastrointestinal tract as a portal of entry. In another set of experiments, Brodie and Elvidge demonstrated that the spread of the poliomyelitis virus to the central nervous system is by way of the nerve tracts rather than by way of the cerebrospinal fluid.

Geiger, Becker and Gray[37] analyzed the records of eighty-four cases of acute poliomyelitis which occurred in San Francisco during May, June, and July of 1934. The percentage of fatalities was 9.5 per cent. Seventy-one per cent of the cases were from four to fifteen years of age. Initial symptoms referable to the nervous system were the most frequent. In thirty-four out of the eighty-four cases, paralysis of one or more types occurred. Paralysis was more common in the sixteen to twenty-five year age group.

Cord Bladder

In a series of 250 cases in which disturbances of bladder function caused by lesions of the nervous system were studied, Lendrum and Moersch[38] found that the diagnosis was central nervous system lues in 106 cases, developmental defect of the caudal end of the spinal cord in fifty-two, arteriosclerosis of the central nervous system in fourteen, and traumatic injury to the spine with or without fracture in thirteen cases. In thirty-two cases various other neurological conditions caused the cord bladder. In thirty-three cases neurologic findings were negative.

Rupture of the Intervertebral Disc

Mixter and Barr[39] report a series of cases of rupture of the disc with herniation of the nucleus pulposus into the spinal canal. These authors point out that this condition should be kept in mind when orthopedic abnormalities of the spine do not respond to appropriate treatment. The symptoms depend on the location and size of the lesion and are similar to those due to any cord tumor. X-ray may show a narrowing of the disc, which is usually significant of migration of the nucleus pulposus, not necessarily but possibly into the spinal canal. Lumbar puncture or combined puncture with lipiodol injection are of aid in diagnosis. A partial or complete block may be demonstrated. Total protein is moderately elevated. By laminectomy the tumor is easily removed unless very small, when it may be very difficult to find.

Peet and Echols[40] also discuss herniation of the nucleus pulposus as a cause of compression of the spinal cord. They report two cases operated on in 1927 and 1928 at which time the tumors removed were considered to be chondromas, but which finally proved to be herniations of the nucleus pulposus.

Neurologic Complications in Pernicious Anemia

Grinker and Kandel[41] studied the effect of liver therapy on the neurologic symptoms in fifty cases of pernicious anemia over a period of several months. They make the following conclusions:

(1.) Liver therapy is not efficacious in improving or preventing degeneration in the central nervous system complicating pernicious anemia.

(2.) Liver improves the general strength of the patient with combined degeneration of the cord by increasing the number of red cells in the circulating blood or by some obscure direct action. Weakness may closely imitate the effects of damage to the spinal cord.

(3.) Liver cures the mental symptoms of pernicious anemia which are usually caused by the anemia.

(4.) The peripheral nerve complications of this disease, consisting of dysesthesia, atrophic changes in the skin, and perhaps other dissociated sensory defects are alleviated by the recession of the anemia. Such complications are much more frequent than they are usually believed to be.

(5.) The majority of cases of combined degeneration of the cord develop rapidly at the onset, they then progress slowly no matter what therapy is employed. Gradual progression of signs may be falsely interpreted as signifying improvement.

(6.) Combined degeneration of the cord develops not infrequently before the anemia and should be diagnosed by the character of the cord syndrome, the achlorhydria, glossitis, and the blood smear.

(7.) Liver therapy in pernicious anemia should be controlled by the height of the blood count. Quantities of liver in excess of that necessary to maintain a normal blood level are wasted.

Syringomyelia

The postoperative results of seventy-eight cases of syringomyelia collected from the literature and including four of his own are recorded by Ley[42]. Immediate results were 68.9 per cent improved, 12.9 per cent unimproved, 18.2 per cent worse (six deaths). Late results (for only sixteen cases) were 64.7 per cent improved, 11.8 per cent unimproved, 23.5 per cent worse. The symptoms responding best to surgical treatment are as follows: objective sen-

sory changes, trophic disturbances, and signs of compression. In some cases there may be improvement in sphincter disturbances. Providing other conditions which may cause a syringomyelic syndrome (syphilis, cervical ribs, vertebral lesions and other external causes) are excluded, the indication for operation is a partial or complete spinal block. In other cases, radiotherapy should be employed. If, however, in these cases there is no improvement after radiotherapy, surgery may be indicated. In serious cases, in which marked trophic disturbances are present or in which rapid progression of symptoms occur, surgical intervention should not be delayed.

NEUROSYPHILIS

Asymptomatic Neurosyphilis

O'Leary[43] points out that asymptomatic neurosyphilis is the forerunner of clinical neurosyphilis and the prevention of the latter type depends on the early treatment of the former type. The diagnosis of asymptomatic neurolues can be made only by examination of the spinal fluid. The increase in the number of lymphocytes and particularly in the number of polymorphonuclear leukocytes means activity of the process and indicates also a more serious type of involvement. A negative spinal fluid, obtained after the second year of primary infection, suggests that clinical neurosyphilis will not develop subsequently. O'Leary states that in 90 per cent of the cases, arsphenamine, bismuth and mercury will control the disease. However, lymphocytosis of the spinal fluid and the presence of the paretic types of colloidal benzoin curve require other measures, such as intraspinal therapy, which is now rarely used, or malaria. These measures will prevent the development of asymptomatic neurolues into clinical neurosyphilis in fifty per cent of the cases in which specific therapy has failed.

The Weather and Neurosyphilis

Petersen[44] has included tabes and paresis in his study of the effect of weather on disease. He associates the meteorological disturbances (infall of cold and polar air) with undue damage to the cord and brain, which is to be found in the region of the storm tracks. Thus, although lues is common in the southern states, tabes and paresis are rare.

Therapy and Neurosyphilis

In a report on twenty-five cases, including various types of neurolues, Spiegel[45] concluded that acetarsone (stovarsol) is a valuable addition to the remedies used in neurosyphilis. In most of the cases observed for seventeen months, there was marked serological improvement in the spinal fluid. The blood Wassermann reaction was, however, less favorably influenced in most cases. There was also marked decrease in cell content, normal counts being obtained after from six to ten injections of acetarsone (.25 or .5 Gm. intravenously once a week). The protein also approached normal figures. No visual disturbances, such as seen after tryparsamide therapy, occurred in any of the cases.

Claude and Masquin[46] make a comparison of the early and late effects of malarial therapy in 327 male cases of paresis treated between 1924 and 1932. An analysis of the immediate effect of the treatment showed: 13.2 per cent greatly improved, 21.2 per cent moderately improved, 28.4 per cent slightly improved, 32.8 per cent unimproved, and 4.4 per cent died in the course of the treatment. Of 260 survivors (1932) there were 23.6 per cent greatly improved, 17.6 per cent moderately improved, 17.0 per cent slightly improved, 18.0 per cent unimproved, 23.8 per cent died in the course of the treatment.

In thirty-two cases of tabes treated with malaria and twenty-four cases treated with arsenic, mercury and bismuth over a period of five years, Yates[47] found that 34 per cent showed improvement by the former therapy and 45 per cent by the use of the latter remedies. Subjective symptoms were used as the basis for evaluating treatment. It was shown that malaria has a definite effect on the spinal fluid cell count and the globulin count. The prognosis was found to be much better when the disease is of shorter duration with a high cell count and globulin, than when the duration is longer with a lower cell count and globulin, whether under malaria or other therapy.

Kuhns[48] gives his experience in the use of the electric blanket for general paresis. Of 230 patients treated by this method at the State Psychopathic Institute of Elgin, Illinois for the past four years, seventy-two per cent were definitely improved; thirty per cent were discharged; and fourteen per cent are on parole. Ten have remained stationary; seven per cent have deteriorated; and eleven per cent have died. With the exception of improvement in speech in eighteen per cent of the cases and in gait in eight per cent, there was little alteration in the other neurological signs. The Wassermann and Kahn tests on the spinal fluid remained the same. The cell count and the colloidal gold test on the spinal fluid often showed marked improvement. Kuhns regards the electric blanket method the safest and simplest form of fever therapy.

CRANIAL AND PERIPHERAL NERVES

Treatment of Trigeminal Neuralgia

Dandy[49] recommends section of the posterior half of the sensory root of the gasserian ganglion for this type of neuralgia. By this procedure not only is the pain abolished, but sensation of the face is unaffected. In 300 operations the neuralgia recurred in only eight cases, probably

due to inconsistency of the arrangement of the pain fibers.

In addition to the trichlorethylene method of treatment for trigeminal neuralgia which relieves many cases, Caldwell[50] recommends the Moyer or castor oil treatment carried out as follows: One or more ounces of castor oil is administered three times a day for several days in succession until the pain is relieved or the treatment judged to have failed. Caldwell states that he has had several remarkable cessations of pain following this treatment.

Dyes[51] reports favorable results in the treatment of trigeminal neuralgia by means of deep Roentgen therapy. Only three out of twenty cases did not respond. Attacks were eliminated for periods varying from three and a half months to eleven years. In eight cases a single radiation or a series extending over several days relieved the attacks. In more chronic cases a repetition of series of treatment was found necessary. The x-ray technique used was 180 kv. (tube), 5 ma., 0.5 mm. Cu., 2.0 mm. Al.

Treatment of Contused Injuries of Peripheral Nerves

Because of his experience with thirty-four cases of injury to nerves contused by such means as fracture, pressure from a cast or tourniquet, Brown[52] recommends early operation. For exploration of the injured nerve in every instance disclosed an intraneural scar between the nerve bundles sufficient to hinder passage of nerve impulses. Expectant treatment with massage, electricity or exercise continued for four to six months is unjustified, Brown feels, in view of the changes occurring in the involved parts during this period, in which the nerve function is absent. Continuous improvement with restoration of function, varying from fifty to one hundred per cent, occurred in Brown's cases. He advises that if no motor or sensory improvement has occurred within one month after injury or if partial involvement tends to become more advanced, operation should be performed.

Facial Palsy

Duel[53] reports very good results in traumatic facial palsy by excising a piece of the injured nerve in the aqueduct and bridging it with a freshly excised nerve graft. Either motor or sensory nerves are used. For filling large gaps the author chooses the anterior femoral nerve as a transplant. Following the operation, there was always a period of muscular inactivity before faradic stimulation elicited a response and then a return of spontaneous movement of the muscles occurred. Final regeneration depended upon whether the injury to the nerve was of recent or of long duration. The author stresses the advantage of immediate exploration of the site of accidental injury of the nerve. In many, an almost perfect recovery will follow removal of a spicule of bone, the lifting of a fractured plate of bone, decompression or cleansing. On the other hand, if extensive damage of the nerve is observed, one should proceed with the transplantation operation. The author also advises early operation in most cases of Bell's palsy; for recovery is much earlier than when the paralysis is allowed to run its course.

Local Anesthesia in the Treatment of Peripheral Nerve Manifestations

Caron[54] states that by the use of local anesthesia many peripheral nerve conditions may be relieved without the necessity of operative procedures. The author has had good results with injections of novocaine, quinine urea hydrochloride, and alcohol in the following conditions: Certain neuritic conditions found in persons with an adipose tendency who, following trauma, develop troublesome tenderness, particularly over the internal aspect of the knees and ankles and the lumbar sacral regions. Spectacular relief may be obtained by injecting a few cubic centimeters of a one per cent solution of quinine urea hydrochloride into the subcutaneous fat. In severe cases of pruritus ani, Caron recommends injections of ten to fifteen cc. of forty per cent ethyl alcohol under the itching area. Ten to twenty mm. of forty per cent alcohol frequently relieves coccydinia. Visceral pain from the heart, gallbladder, kidney, pelvis, or pain from other intra-abdominal origin can be temporarily relieved by subcutaneous infiltration of novocaine. For the pain of laryngeal tuberculosis, Caron uses two cc. of a ninety-five per cent alcohol injected into the superior laryngeal nerve on each side. In six of every ten cases of sciatica, the author has had good results from single injections of one per cent quinine urea hydrochloride into the sheath of the sciatic nerve.

MUSCULAR DYSTROPHY AND MYASTHENIA GRAVIS

Chemistry

Nevin[55], in his chemical studies of muscles, found that the phosphorus-holding compounds which play an important rôle in the chemistry necessary for muscular contraction were normal in myasthenia, whereas in pseudohypertrophic muscular dystrophy and in myotonia some alterations from the normal were found. In myotonia the total acid-soluble phosphorus was found to be below normal and the creatine phosphoric acid content slightly diminished. In contrast to the view held by Remen (who has observed improvement in myasthenia from glycine therapy) that in this disease creatine-creatinine metabolism is primarily disturbed, Nevin holds to the theory that such disturbances in metabolism are secondary effects of the disease.

Therapy

Kostakow[56], studying three generations of a family comprising fifty-five persons, forty-seven of whom are living, found that fifteen of them developed muscular dystrophy. These were all males, the females acting only as transmitters. The author has treated seventeen cases with glycocoll (glycine). Two cases, treated for two months showed no improvement; three were able to walk five km. without tiring; seven were able to walk ten km. without tiring; four were able to walk fifteen km. without tiring. The degree of improvement was found to be in direct ratio to the muscle volume still present and in inverse ratio to the duration and degree of involvement of the disease. The effect of medication depended in particular on the length of time it was given without interruption.

Tripoli and Beard's[57] experience with glycine and glutamic acid treatment for progressive muscular atrophy and progressive muscular dystrophy was found to coincide with that of other authors. Of sixty-nine cases collected from the literature by these authors, clinical improvement has been recorded in fifty-one cases.

Reinhold, Clark, Kingsley, Custer and McConnell[58] found that glycine feeding in nine cases of progressive muscular dystrophy caused a definite regeneration of muscle as disclosed by biopsy. Clinical improvement, however, did not appear to coincide with the change in the composition of the muscle. Better clinical results were noted in children than in adults. One child, after three months of treatment, regained the ability to stand for brief intervals when supported. Contractures of the hamstring muscles were ameliorated in another child, after a similar period of treatment. Although muscle function was not very encouraging, there was noted, particularly in children, improvement in mental and physical activity.

Cuthbertson and Maclachlan[59] treated nine cases of various types of muscular dystrophy, most of which showed improvement in the power of some of the specific muscles affected. In two cases of muscular atrophy, some general improvement was observed. The authors believe that in most cases the changes noted were greater than might be expected to occur from hospitalization alone.

Effects of Lightning and Electricity on the Central Nervous System

Critchley[60] discusses the neurological manifestations of electrical injuries and lightning stroke. Pathologically, the main effects were found to be: (a) focal petechial hemorrhages scattered throughout the brain, especially in the medulla; and in the spinal cord, the anterior horn cells were particularly involved; (b) chromatolysis, especially in the pyramidal cells, in the cells in the medullary nuclei, in the anterior horn cells and in the Purkinje cells of the cerebellum; (c) wide dilatation of the perivascular spaces, especially in the brainstem and cervical cord; (d) fragmentation and tortuosity of peripheral nerve axons with breaking down of the sheath of Schwann and infiltration of the epineurium with endothelial cells; (e) with unusually severe injury, the entire brain and parts of the cord are found to be swollen, softened, and even diffluent.

Clinically, pain may be agonizing; unconsciousness may occur either from syncope or from immediate concussion. Late unconsciousness may result from cerebral edema. Deafness and visual disorders are common. Severe electrical injuries may cause great restlessness, irritability, and convulsions. Later effects may include: (1) the syndrome of headache, dizziness, insomnia, and forgetfulness; (2) spinal atrophic paralysis in certain muscle groups; (3) diffuse involvement called "electrotraumatic encephalomyelitis"; (4) peripheral nerve lesions; and (5) various psychoses.

REFERENCES

1. Brodie, M., and Elvidge, A. P.: Portal of entry and transmission of virus of poliomyelitis. Science **79:** 235 (March 9) 1934.
2. Petersen, W. F.: The Patient and the Weather. Volume I. Part I. Ann Arbor: Edwards Brothers, Inc. 1935.
3. Gibbs, F. A.; Lennox, W. G., and Gibbs, E. L.: Cerebral blood flow preceding and accompanying epileptic seizures in man. Arch. Neurol. & Psychiat. **32:** 257 (Aug.) 1934.
4. Finesinger, J., and Cobb, S.: Cerebral circulation; action on pial arteries of the convulsants caffeine, absinth, camphor and picrotoxin. Arch. Neurol. & Psychiat. **30:** 980 (Nov.) 1933.
5. Engel, R.; McQuarrie, I., and Ziegler, M.: Mineralhaushaltsstudien bei jugendlichen Epileptikern. Arch. f. exper. Path. u. Pharmakol. **174:** 555 (March 13) 1934.
6. Jacobsen, A. W.: Pitressin test in epilepsy. New York State J. Med. **34:** 506 (June 1) 1934.
7. Paskind, H. A.: Relationship of migraine, epilepsy and some other neuropsychiatric disorders. Arch. Neurol. & Psychiat. **32:** 45 (July) 1934.
8. Lennox, W. G.: Use of ergotamine tartrate in migraine. New Eng. J. Med. **210:** 1061 (May 17) 1934.
9. Lennox, W. G.; Gibbs, E. L., and Gibbs, F. A.: Effect of ergotamine tartrate on cerebral circulation of man. J. Pharmacol. & Exper. Therap. **53:** 113 (Jan.) 1935.
10. Brock, S.; O'Sullivan, M., and Young, D.: Effect of nonsedative drugs and other measures in migraine, with especial reference to ergotamin tartrate. Am. J. M. Sc. **188:** 253 (Aug.) 1934.
11. Logan, A. H., and Allen, E. V.: Treatment of migraine with ergotamine tartrate. Proc. Staff Meet. Mayo Clin. **9:** 585 (Sept. 26) 1934.
12. Geiger, A. J.: Purpura haemorrhagica with cerebrospinal hemorrhage; report of 2 cases. J. A. M. A. **102:** 1000 (March 21) 1934.
13. Diamond, I. B.: Leukemic changes in brain; report of 14 cases. Arch. Neurol. & Psychiat. **32:** 118 (July) 1934.
14. Leary, T.: Subdural hemorrhages. J. A. M. A. **103:** 897 (Sept. 22) 1934.
15. Munro, D.: Diagnosis and treatment of subdural hematomata; report of 62 cases. New Eng. J. Med. **210:** 1145 (May 31) 1934.
16. Lyerly, J. G.: Management of acute craniocerebral injuries. South. Med. & Surg. **96:** 326 (July) 1934.
17. Russell, W. R.: After-effects of head injury. Tr. Med.-Chir. Soc. Edinburgh. 1933-1934. p. 129; in Edinburgh M. J. (Sept.) 1934.
18. Dyke, C. G., and Davidoff, L. M.: Recent advances in encephalography. Radiology **22:** 461 (April) 1934.
19. Masserman, J. H.: Intracranial hydrodynamics; central nervous system shock and edema following rapid fluid decompression of ventriculo-subarachnoid spaces. J. Nerv. & Ment. Dis. **80:** 138 (Aug.) 1934.
20. Muckenfuss, R. S.; Armstrong, C., and Webster, L. T.: Etiology of 1933 epidemic of encephalitis. J. A. M. A. **103:** 731 (Sept. 8) 1934.
21. Webster, L. T., and Fite, G. L.: Virus encountered in study of material from cases of encephalitis in St. Louis and Kansas City epidemics of 1933. Science **78:** 463 (Nov. 17) 1933.

22. Gay, F. P., and Holden, M.: Further evidence for presumed herpetic origin of epidemic encephalitis. Tr. A. Am. Physicians **48:** 16, 1933.
23. Leake, J. P.; Musson, E. K., and Chope, H. D.: Epidemiology of epidemic encephalitis, St. Louis type. J. A. M. A. **103:** 728 (Sept. 8) 1934.
24. McCordock, H. A.; Collier, W., and Gray, S. H.: Pathologic changes of St. Louis type of acute encephalitis. J. A. M. A. **103:** 822 (Sept. 15) 1934.
25. Neal, J. B.: Recent advances in treatment of epidemic encephalitis. New York State J. Med. **34:** 707 (Aug. 15) 1934.
26. Mutch, N.: Medicinal treatment of chorea; calcium aspirin. Brit. M. J. **2:** 246 (Aug. 11) 1934.
27. Sauter, E.: Zum Schicksal der Encephalitiker. Katamnestische Untersuchungen der an akuter Encephalitis lethargica erkrankten und an der Zürcher medizinischen Klinik hospitalisierten Patienten. Schweiz. med. Wchnschr. **64:** 464 (May 19) 1934.
28. Weinberg, M. H.: Lecithin treatment of some multiple sclerosis syndromes. J. Nerv. & Ment. Dis. **79:** 264 (March) 1934.
29. Neymann, C. A., and Osborne, S. L.: Treatment of some multiple scleroses by electropyrexia. J. Nerv. & Ment. Dis. **79:** 423 (April) 1934.
30. Abramson, J. L.: Acute lymphocytic meningitis. Arch. Neurol. & Psychiat. **31:** 1235 (June) 1934.
31. Viets, H. R., and Watts, J. W.: Acute aseptic meningitis J. Nerv. & Ment. Dis. **80:** 253 (Sept.) 1934.
32. Felsen, J., and Osofsky, A. G.: Streptococcus (viridans) meningitis with recovery; immunologic studies. J. A. M. A. **102:** 2170 (June 30) 1934.
33. Chopra, B. L.: Case of cerebro-spinal fever successfully treated by intravenous injections of urotropine. Indian M. Gaz. **69:** 263 (May) 1934.
34. Bedell, C. C.: Pneumococcic meningitis; report of case with recovery following cisternal drainage. J. A. M. A. **102:** 820 (March 17) 1934.
35. Reveno, W. S., and McLaughlin, N.: Pneumococcic meningitis; recovery with Felton's serum. Ann. Int. Med. **7:** 1026 (Feb.) 1934.
36. Parry, C. L.: Tuberculous meningitis with recovery. Brit. M. J. **2:** 859 (Nov. 10) 1934.
37. Geiger, J. C.; Becker, G. H., and Gray, J. P.: Acute anterior poliomyelitis; report on first phase of 1934 outbreak. J. A. M. A. **103:** 342 (Aug. 4) 1934.
38. Leudrum, F. C., and Moerach, F. P.: Neurologic diagnosis in 250 cases of cord bladder. J. A. M. A. **102:** 658 (March 3) 1934.
39. Mixter, W. J., and Barr, J. S.: Rupture of intervertebral disc with involvement of spinal canal. New Eng. J. Med. **211:** 210 (Aug. 2) 1934.
40. Peet, M. M., and Echols, D. H.: Herniation of nucleus pulposus; cause of compression of spinal cord. Arch. Neurol. & Psychiat. **32:** 924 (Nov.) 1934.
41. Grinker, R. R. and Kandel, E.: Pernicious anemia; results of treatment of neurologic complications. Arch. Int. Med. **54:** 851 (Dec.) 1934.
42. Ley, A.: El tratamiento quirurgico de la siringomielia. Rev. de cir. de Barcelona **7:** 161 (March) 1934.
43. O'Leary, P. A.: Asymptomatic neurosyphilis. Proc. Staff Meet. Mayo Clin. **9:** 756 (Dec. 12) 1934.
44. Petersen, W. F.: Significance of American distribution of tabes and paresis. Am. J. Syph. & Neurol. **18:** 75 (Jan.) 1934.
45. Spiegel, L.: Treatment of neurosyphilis with acetarsone (stovarsol) given intravenously; preliminary report. Am. J. Syph. & Neurol. **18:** 56 (Jan.) 1934.
46. Claude, H., et Masquin, P.: Le devenir des paralytiques généraux malarisés; expérience de neuf ans de malariathérapie. Presse méd. **41:** 2005 (Dec. 13) 1933.
47. Yates, A. G.: Some observations on treatment of tabes with malaria. Lancet **2:** 1335 (Dec.) 1934.
48. Kuhns, R. H.: Treatment of general paresis by electropyrexia. Arch. Phys. Therapy **15:** 725 (Dec.) 1934.
49. Dandy, W. E.: Diagnosis and treatment of lesions of cranial nerves. Delaware State M. J. **6:** 153 (July) 1934.
50. Caldwell, J. A.: Trigeminal neuralgia and other painful conditions about face. J. Med. **15:** 7 (March) 1934.
51. Dyes, O.: Ueber die Behandlung der Trigeminusneuralgie. München. med. Wchnschr. **81:** 1603 (Oct. 19) 1934.
52. Brown, H. A.: Contused injuries of peripheral nerves—value of early surgical treatment. California & West. Med. **41:** 166 (Sept.) 1934.
53. Duel, A. B.: Operative treatment of facial palsy. Brit. M. J. **2:** 1027 (Dec. 8) 1934.
54. Caron, R. P.: Application of principles of local anaesthesia to treatment of peripheral nerve manifestations. Am. J. Surg. **23:** 521 (March) 1934.
55. Nevin, S.: Study of muscle chemistry in myasthenia gravis, pseudohypertrophic muscular dystrophy and myotonia. Brain **57:** 239 (Oct.) 1934.
56. Kostakow, S.: Die progressive Muskeldystrophie, ihre Vererbung und Glykokollbehandlung. Klin. Wchnschr. **13:** 219 (Feb. 10) 1934.
57. Tripoli, C. J., and Beard, H. H.: Muscular dystrophy and atrophy; clinical and biochemical results following oral administration of amino-acids. Arch. Int. Med. **53:** 435 (March) 1934.
58. Reinhold, J. G.; Clark, J. H.; Kingsley, G. R.; Custer, R. P., and McConnell, J. W.: The effects of glycine (glycocoll) in muscular dystrophy. J. A. M. A. **102:** 261 (Jan. 27) 1934.
59. Cuthbertson, D. P., and Maclachlan, T. K.: Treatment of muscular dystrophy with glycine. Quart. J. Med. **3:** 411 (July) 1934.
60. Critchley, M.: Neurological effects of lightning and of electricity. Lancet **1:** 68 (Jan. 13) 1934.

THE NEXT STEP IN CANCER CONTROL

In the November Bulletin of the American Society for the Control of Cancer, the announcement of the plans for the instruction of the laity appears in these words:

"The Society, therefore, proposes to turn the major portion of its energies, for the present, to a widespread and intensive campaign for informing the laity of the known facts concerning the prevention of cancer and the possible signs which may mean the presence of that condition or of a precancerous lesion.

"It hopes and expects to enlist the services of organized medicine in every community to help it to keep the information given both accurate and up-to-date. It hopes to interest many thousands of persons in obtaining frequent complete physical examinations and in relying on the profession to advise them wisely as to treatment.

"It hopes and expects also that the medical profession will constantly bear in mind the extremely conservative attitude and continual reliance in the profession which our Society has shown. As a result of this the Society believes that the profession will completely understand and will cordially support in every possible way, the program of nation-wide lay education which the Society now adopts.

"Cancer mortality can be cut. Ample clinical data support this conclusion. The profession will benefit by an organized lay effort in this direction. The cancer patient will be given the best possible chance for survival and for decreased suffering. The slogan—long in use by our Society — 'Fight Cancer with Knowledge' is about to be spread to millions now in ignorance of its value and truth. In this effort all should find a worthwhile outlet for humanitarian impulses. Within a few weeks more complete information of the general program will be made public. In the meanwhile volunteers both from professional and lay ranks are needed in the fight."

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 21511

PRESENTATION OF CASE

A forty-five year old white Italian housewife was admitted complaining of pain in the head, and stiff neck.

Two days before entry the patient was suddenly seized with pain in the head, dizziness, and stiffness of the neck. She did not fall but required assistance to her bed. The pain in the head and neck persisted and she vomited several times. She became drowsy, then semiconscious and remained in this state until her admission.

The patient had been moderately dyspneic after exertion for the past eight years. This had gradually become more marked during the two years preceding her admission. She had occasional epistaxis and her vision seemed to be failing gradually. For about three weeks before admission she had generalized headache of moderate severity. During this time she suffered a gripping pain over the heart which recurred approximately every three days, lasted for about five minutes, and subsided spontaneously. There was no radiation. Causative factors were not recorded. She had frequent gaseous eructations following these episodes and occasionally vomited. Her diurnal urinary frequency had increased and there was a nocturia of two to three times. For about one year she had noticed a mass in her left lower quadrant which had gradually increased in size. Her abdomen occasionally became so large that she was unable to wear corsets. Her catamenia were normal.

The patient had been admitted to this hospital five years previously for pain in the chest and fever, following an acute upper respiratory infection which had begun eight weeks before. Pleural fluid removed at this time was thought to be consistent with tuberculosis although guinea pig inoculation and culture were negative. She had a febrile course and was discharged to a sanitarium after one month in the hospital. She remained in the sanitarium for eight months and left there much improved. Since that time she had no symptoms of respiratory disease. Because of hypertension (195/115), which had been recognized at her previous admission, her activities were restricted and she had adhered to a diet which caused the loss of thirty pounds.

Physical examination showed a rather obese, semicomatose woman. The neck was rigid. The fundi showed notching of the veins with slight thickening and tortuosity of the arterioles. The discs were normal. The heart was slightly enlarged to the left and the blood pressure was said to be high although no figures were recorded. The abdomen was distended and there was a hard, rounded, freely movable mass believed to be attached to the fundus of the uterus in the left lower quadrant. Bilateral positive Hoffman signs and left Babinski were elicited. The tendon reflexes were found to be more active on the left side.

The temperature was 95.5°, the pulse 60. The respirations were 25.

Examination of the urine showed a specific gravity of 1.028. There was a trace of albumin and a Benedict's test produced a green precipitate. The sediment was loaded with red blood cells and there were a few white blood cells. No casts were seen. The blood showed a red cell count of 4,350,000, with a hemoglobin of 90 per cent. The white cell count was 7,200, 87 per cent polymorphonuclears. A stool examination was negative. The nonprotein nitrogen of the blood was 50. A lumbar puncture showed an initial pressure of 500 millimeters and a final pressure of 200 millimeters. The cell count of spinal fluid showed 600,000 cells per cubic millimeter, practically all of which were red blood cells. An ammonium sulphate test was positive. The total protein was 261 milligrams. A spinal fluid Wassermann examination was negative.

The patient received five daily lumbar punctures, each of which showed decreasing amounts of blood. The pressure likewise diminished. On the second day the patient regained full consciousness. She improved generally but still had a severe headache and a stiff neck. She was discharged on the eighteenth day.

Final Admission, twelve days later.

After her discharge the patient restricted her activity and felt fairly well. Four days prior to reëntry she developed constant severe pain in the lower abdomen associated with nausea and repeated emesis. Her last normal bowel movement occurred three days before entry and she passed neither stool nor gas thereafter despite the administration of enemata.

Physical examination showed the patient to be quite ill and complaining of severe abdominal pain which was more tolerable when she sat upright. The skin of the extremities was cold and clammy. There was profuse perspiration. The pupils were constricted, equal, and did not re-

act to light. The mucous membranes of the mouth were dry and red. The breath was foul. The heart was unchanged except for a pulse of 110. The blood pressure was 100/80. The lungs were clear. The abdomen was full and distended. Audible peristalsis was absent. No fluid wave was elicited. There was generalized tenderness with deep pressure and voluntary muscle spasm. In the lower abdomen a smooth, readily movable pumpkin-sized tumor was palpated, lying more on the right side. Pelvic examination showed a moderately lacerated high cervix. The fundus was not felt. The mass appeared to be separate from the uterus. The vaults were otherwise clear.

The temperature was 99.6°. The respirations were 20.

Examination of the urine showed a specific gravity of 1.030 with a trace of albumin. The blood showed a white cell count of 20,000, with a hemoglobin of 90 per cent. A lumbar puncture showed clear fluid with an initial pressure of 140 and a final pressure of 65. The nonprotein nitrogen of the blood was 77.

By means of a gastric tube about twenty ounces of brown fluid with a foul odor was removed from the stomach. The patient was treated supportively by intravenous fluids, morphin, the application of warmth, and constant duodenal drainage. Her general condition improved slightly but shortly thereafter the blood pressure dropped to 68/54, the pulse rose to 130, the temperature to 107°, and she expired about thirty-six hours after reëntry.

Differential Diagnosis

Dr. Edward L. Young, Jr.: The past history gives the suggestion of hypertension and probably some accompanying chronic nephritis and the story of a mass in the lower abdomen which has gradually increased in size. Five years before that there was a story of prolonged upper respiratory infection with pleurisy and the belief that it was tuberculosis, so that she was put in a sanitarium for eight months and at that time she had a hypertension of 195/115. Following a diet she lost thirty pounds in weight.

"The urine showed a specific gravity of 1.028." That is rather high for chronic nephritis of any degree, although such a gravity might be explained by the presence of sugar, and there is a positive Benedict test.

It seems to me that the essential part of the picture on the first admission is a patient with hypertension and evidence of a cerebral accident.

"Four days prior to reëntry she developed constant severe pain in the lower abdomen associated with nausea and repeated emesis." I think the time element may be important there.

"Her last normal bowel movement occurred three days before entry and she passed neither stool nor gas thereafter despite the administration of enemata." I call attention to that because it is entirely possible that a carcinoma of the large bowel is present which has given no symptoms at all previously and has suddenly shut down. If that is true there would not be immediate vomiting. Obstruction of the large bowel is almost invariably followed by vomiting, not at once but two or three days later, and it is not an early prominent symptom. The immediate vomiting suggests a rather more serious accident than that and suggests that the blood supply of bowel is immediately compromised.

"The urine showed a specific gravity of 1.030 with a trace of albumin." Again suggesting the possibility of sugar; or if her bowels did not move she may have been taking magnesium sulphate and occasionally we get a high specific gravity of urine following magnesium sulphate where for some reason it is absorbed.

Her condition on admission was altogether too poor to justify any surgical interference, even if that were considered indicated at the moment.

There are two or three features about this case that I think we must mention, either to consider relevant or to rule out. First, is the tumor important? They say it is a tumor the size of a pumpkin. Of course there are various sizes of pumpkins but it does suggest a pretty large tumor. A malignant tumor of the ovary seldom gets to that size without causing more trouble from malignancy and more fluid. There was not enough fluid in the abdomen to be able to detect; in any event if it is malignancy from outside there should be evidence of carcinoma and, it seems to me, a slower shutting down of the obstruction. This was a very sudden catastrophe that she had. Secondly, is it possible that this is a chronic nephritis and that we are here seeing what we do see occasionally, the terminal stage of renal insufficiency? We do see those cases in which the abdominal symptoms are prominent, and it often happens that operation is undertaken in some of these cases for supposed acute abdominal emergency. It seems to me that we do not have sufficient evidence for a chronic nephritis of that severity to justify the belief that that is a probable diagnosis. That brings us back to the possible cause of an acute intestinal obstruction of sufficient severity so that immediate nausea and vomiting take place and the patient goes into shock. She was in shock on admission and died within thirty-six hours. Is it possible that her cardiac condition was such that it could account both for the cerebral accident four weeks before and for the present emergency presumably through embolism to a mesenteric artery? It is characteristic of mesenteric thrombosis that there is severe pain, that there is absent peristalsis at

the end of a relatively short time and that there may be perforation. The shock of a mesenteric embolus—I believe this sudden onset would be embolism rather than thrombosis—plus a perforation would have resulted in immediate death. Or did she suffer one of the other possible causes of sudden upper abdominal obstruction, an acute volvulus or a band, both of which involved the small bowel and the mesentery and would result in the severe picture that she has? I have never made a diagnosis correctly before operation of mesenteric embolism or thrombosis and because I have everything to gain and nothing to lose I am going to say that that seems like the best bet to me, with volvulus second. I do not think the condition is connected with the tumor and I do not believe it is the end stage of a chronic nephritis.

Dr. J. H. Means: I think the kind of cerebral episode is of some interest. The description here is almost the classical one of spontaneous subarachnoid hemorrhage. The patient was hypertensive and I suppose that the more usual kind of cerebral episode with hypertension is an ordinary form of apoplexy, not the so-called subarachnoid hemorrhage. I wonder if this may be the clue to some strange diagnosis in this case. I cannot suggest what it might be. I should be very much interested to find out what kind of cerebral hemorrhage it was. The picture is more that of subarachnoid hemorrhage than apoplexy with hemorrhage into the ventricle. I think the recovery is more in favor of the former.

A Physician: Is there a possibility of there being a dissecting aneurysm there?

Dr. Means: Dissecting aneurysm is a very good thing to have on the list of any differential diagnosis of cause of sudden death.

Clinical Diagnoses

Tumor of the ovary.
Intestinal obstruction.

Dr. Edward L. Young's Diagnoses

Acute intestinal obstruction with vascular damage.
1. Mesenteric thrombosis.
2. Volvulus.

Anatomic Diagnoses

Leiomyoma of the left Fallopian tube.
Volvulus of the small intestine, with obstruction.
Bronchopneumonia, bilateral.
Hemorrhage of the cerebellum, right.
Infarct of the left putamen.
Arteriosclerosis, moderate; aortic and cerebral.

Pathologic Discussion

Dr. Tracy B. Mallory: The findings at autopsy show that the large tumor which she had almost certainly had for many years, although it was not noticed on the first entry, was a fibroid loosely attached to the Fallopian tube, not in the ovary or the uterus. It had picked up a blood supply from the omentum and from an adherent loop of ileum. It had then twisted on its stalk three times and carried the ileum around with it, producing a volvulus and complete intestinal obstruction. The gut was widely dilated above the point of twisting, and it had collapsed below.

She showed in addition the usual findings of hypertension, a hypertrophied heart, slightly atrophic kidneys and diffuse arteriolarsclerosis of the kidneys. There were no thrombi in the cardiac cavities to serve as sources of emboli. The lungs showed a terminal bronchopneumonia.

At the time of autopsy there was no longer any trace of subarachnoid hemorrhage though the history leaves no doubt that it must have been present on her previous entry. In view of the story Dr. Kubik made a very careful search for a congenital aneurysm of the circle of Willis but could not find one. He did find a fairly fresh area of infarction and hemorrhage in the cerebellum and a much older softening in the left putamen. The cerebellar lesion was in all probability the source of the subarachnoid hemorrhage.

Dr. Young: Why was not the tumor sensitive? That is why I threw out the question of twist. It says "a freely movable tumor" and nothing is said about sensitiveness.

Dr. Mallory: I do not believe we can be sure what they were feeling on the last entry; it may have been the strangulated loop of bowel rather than the neoplasm itself.

CASE 21512

Presentation of Case

First Admission. A twenty-seven year old American shoe factory operator entered complaining of severe abdominal pain.

During the past month he had had "indigestion" off and on, characterized by mild attacks of cramplike abdominal pain. The pain was not associated with meals until one week before entry, when it began to occur about fifteen minutes after each meal and last for about half an hour. His bowels had been regular. There were no additional symptoms until three days before entry, when he began to have sharp, cramplike, severe, non-referred pain in the upper abdomen which continued until admission without relief. He had been unable to sleep since the onset of the pain. He was slightly nauseated two or three times on the evening be-

fore admission but did not vomit. His bowels had moved on the day of admission as usual.

There was no history of jaundice, hematemesis or tarry stools. There was no loss in weight. For several years he had had nocturia about once a night but during the past week he voided about three times a night.

Physical examination was negative except for the abdomen. In the right upper quadrant there was a rounded, fairly firm, quite tender, freely movable mass which was not attached to the liver and which could be pushed into the right or left lower quadrant with ease. There was slight spasm over the mass. The liver, spleen and kidneys were not felt.

The temperature, pulse and respirations were normal.

A Wassermann test was moderately positive.

On the day of admission an exploratory laparotomy was performed. When the greater omentum was turned back a somewhat lobulated mass approximately 15 by 8 by 6 centimeters was found involving the mesentery of the jejunum. The mass was cystic for the most part, but in places was firm and in other places definitely whitish in color and extended to the duodenojejunal juncture, making resection very difficult. A jejunostomy was performed short-circuiting the mass, which was left intact. He was discharged on the thirteenth day.

Second Admission, ten years later.

He had been perfectly well and symptom-free during this period. Five days before this entry, after lunch, he was suddenly seized with a fairly severe right upper quadrant pain exactly similar to that experienced before his previous admission. The pain continued unaltered until admission. It was steady and constant but not associated with nausea or vomiting.

Physical examination showed a localized round area of spasm and tenderness in the right upper quadrant but a definite mass could not be felt.

Two days later an exploratory laparotomy was performed and the tumor removed. He had an uneventful convalescence and was discharged on the eleventh day.

Differential Diagnosis

Dr. John Stewart: We seem to have here a definite picture of rather unlocalized, frequent upper abdominal pains of peristaltic origin, and our attention is at once focused on the small intestine. The severity of the pain in the absence of vomiting is interesting. Ordinarily one expects to see vomiting associated with such severe peristaltic pain, arising in the small intestine, if the lesion is obstructive in type. The fact that obstipation was present is further evidence that we are not dealing with one of the usual types of intestinal obstruction.

An important observation in the physical examination is the mobility of the mass described. We infer that the mass either had a pedicle of some length or else was attached to a mobile viscus, such as the great omentum, small intestine, or mesentery.

The fact that operation was performed so soon is interesting in considering the symptomatology. From the evidence at hand one hardly sees a pressing indication for immediate operation without further study. There is described a mass apparently fairly well circumscribed lying in the mesentery of the jejunum and evidently interfering with jejunal peristalsis. The mass is at least partially cystic, and the meager details concerning its physical nature bear out the presumption already established of absence of active acute inflammation.

To be considered in the differential diagnosis are the following: first, a chronic inflammatory mass, such as gumma or mass of tuberculous lymph nodes; secondly, a benign neoplasm, such as lipoma, fibroma or neurofibroma; thirdly, a malignant tumor, such as fibrosarcoma, lymphosarcoma, or one of the rarer tumors arising in tissue of the sympathetic nervous system, including paraganglioma and ganglioneuroma; fourthly, a mass arising in connection with misplaced tissue, possibly of pancreatic or splenic origin, in which group also might be placed the so-called mesenteric cyst; finally, the mass may be springing from the pancreas and presenting in the root of the intestinal mesentery.

The subsequent course in this case after the first operation is of much significance in the differential diagnosis. A malignant tumor is no longer to be considered, for not only has the mass not produced symptoms or increased in size during ten years, but the lesion can now be excised. The same considerations make a chronic inflammatory mass an unlikely possibility. Besides, if the lesion were tuberculous there should be other obvious tuberculous nodes noted in the findings at operation. It seems unlikely that the lesion arose in the pancreas itself, for it seems to be too freely movable. From the description of the findings at the first operation and from the statement about the second operation, one assumes that the lesion does not spring from the bowel itself, for we are told that the tumor was removed, apparently without resection of the intestine. The diagnosis, then, seems to involve a benign, at least partially cystic mass in the jejunal mesentery. Lipoma and fibrous tumor cannot be excluded but are unlikely. The most plausible diagnosis in the light of the facts given us seems to be mesenteric cyst. At any rate, this is certainly an unusual lesion.

Preoperative Diagnosis

Tabes mesenterica.

Dr. John Stewart's Diagnosis

Mesenteric cyst.

Pathologic Diagnosis

Lymphangioma of the mesentery.

Pathologic Discussion

Dr. Tracy B. Mallory: This patient was operated on for the second time by Dr. Vincent with a preoperative diagnosis of probable tuberculosis of the mesenteric lymph nodes. When he got into the abdomen he found a tumor mass about the size of a baseball situated in the mesentery of a high loop of jejunum. It was adherent along one surface to the intestine, and on the opposite side it extended deeply into the root of the mesentery. It felt partly firm and partly cystic. He was able to dig it out from its attachment in the retroperitoneal tissues and resected it along with about a foot of the adherent jejunum. The patient made an uneventful recovery.

The specimen which we received in the laboratory showed a well-encapsulated tumor mass 9 by 5 by 4.5 centimeters. On section it was found to consist of innumerable cavities, varying in size from bare visibility to two centimeters in diameter. Many of the cysts contained creamy chylous material; others showed all grades of blood staining up to frankly hemorrhagic content.

Microscopic examination showed that it was made up of a mass of anastomosing channels lined by endothelium, the majority of which contained partially coagulated protein material. In many places well-formed lymphatics with connective tissue and smooth muscle in their walls can be made out. In other parts of the tumor the formation of lymphoid follicles and even of small lymph nodes is apparent.

This tumor is a characteristic example of the lymphangiomas of the mesentery. They probably arise in the region of the peritoneal tissues. Some of them tend to invade backward and downward along the aorta or about the kidneys. Others, like this one, grow forward into the mesentery and eventually come in contact with a loop of small intestine. The formation of nodules of lymphoid tissue in the tumors has frequently been described and is apparently fairly typical of it. The condition is closely analogous to the so-called hygroma colli, which is really a cavernous angioma of the neck. Sometimes the tumors grow much more rapidly than this one apparently did, but they evidently have little tendency toward metastasis.

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IMPORTANCE OF THE PHYSICIAN IN A HEALTH PROGRAM

WHEN health was largely the concern of individuals not organized in groups, it was the persons who were sick and the healers who carried the chief responsibilities. As the significance of disease to the community came to fuller realization, organizations of lay persons as well as of physicians developed, until, at present, with the enormous widening of lay interest, physicians may be said to constitute a minority group. There has been also a strong tendency for lay groups to tell physicians how they should practice medicine.

Nevertheless, the keystone of the arch is the physician, and it is of critical importance to the community that the physician shall receive the best kind of preparation for the exacting office he is expected to perform. To say that he shall be socially minded does not mean that he shall be a person of slight individuality: it means simply that he shall see the problems presented by the individual patient against the background of a group, a society, of which the patient is a member. The wider implications of a social organism of which an individual part is sick must be taken into consideration.

With the growing realization of the complexities of civilized life, and underneath the realization that life is actually becoming more complicated, we are faced with the question of whether the minds of men can deal with the added burden. Are mental breakdowns due to unwillingness or incapacity to face the facts and to make the necessary adjustments?

The demands of advancing civilization are such that of the physician as well as of others, but especially of the practitioner of medicine, there is required mental flexibility and adaptability, toughness and resiliency of mind. The physician occupies one of the key positions in advancing civilization. Nothing less than the best can be expected of him and so nothing less than the best must be given him.

The preparation of the physician for his exacting duties must be the best preparation. About the details we may disagree, but the quality must be excellent. It must excel the ordinary, the mediocre, in thoroughness, in comprehensiveness, in adequacy and in appropriateness. It must have distinction.

It is on this account that the formal standards set by the state for admission to the practice of medicine shall be changed from time to time to keep step with the practice of medicine. Science must and will lead the way. Education brings the gifts of science to practice. If education is antiquated, defective, substandard, the patient pays, sometimes with his life.

There is a popular delusion, often apparent in political circles, though not unknown elsewhere, that information is identical with education. Translated into practical affairs, this means that a candidate who manages to acquire enough information to get a passing mark on examination is therefore supposed to be qualified for practice. But in fact the correlation between information and education is not close enough for the presence of the first element to be accepted as a guarantee of the presence of the second.

Education, the best possible education, of physicians is demanded for the welfare of the citizens of the Commonwealth. The state has educational standards. They should be applied rigorously. The chartering of a medical school by the state should mean, not that the institution is free to carry on its work in utter disregard of the principles of education, but that the state guarantees a reasonable approximation to generally accepted standards of education. Is this too much to expect? We think not. Furthermore it is the right of the citizens to demand of the government that it protect them adequately against substandard physicians, graduates of substandard schools.

Christmas

We are entering upon another season; another year is about to roll to its close, and fittingly enough the beginning of the sun's return to northern latitudes is marked by the most beloved and the kindliest of our religious festivals. It is pleasant to believe that at the Christmas time, when a Saviour was born to bring spiritual salvation to an oppressed people, we also may see the effulgent glow of an imaginary Star, marking a goal and setting an ideal toward which we may at least strive with a renewal of that hope so necessary for the success of man's efforts.

Fortunate we are in belonging to a profession which, imperfect as it may be, susceptible as it may be to the frailties of human nature, nevertheless has traditionally given of itself to a degree which sets it apart as a particular and a uniquely honorable Estate. That season of the year has come which has been immemorially dedicated to the giving and the receiving of gifts.

Let our choicest gifts this year be part of ourselves, as we remember that the gift without the giver is bare.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Faulkner, James M. A.B., M.D. Harvard University Medical School 1924. Assistant in Medicine, Harvard University Medical School. Research Fellow, Thorndike Memorial Laboratory. Junior Visiting Physician, Boston City Hospital. Visiting Physician, House of the Good Samaritan. His subject is "The Electrocardiographic Diagnosis of Acute Cardiac Infarction with Special Reference to the Value of Precordial Leads." Page 1215. Address: 264 Beacon Street, Boston.

Young, Edward L., Jr. A.B., M.D. Harvard University Medical School 1909. F.A.C.S. Assistant Visiting Surgeon, Massachusetts General Hospital. Surgeon-in-Chief, Faulkner Hospital. His subject is "Secondary Carcinomata of the Large Bowel." Page 1219. Address: 279 Clarendon Street, Boston.

Muller, Gulli Lindh. A.B., M.D. College of Physicians and Surgeons, Columbia University 1921. Formerly, Assistant Physician, Thorndike Memorial Laboratory, Boston City Hospital. Research Fellow in Medicine, Harvard University Medical School. Now, Pathologist, Rutland State Sanatorium. His subject is " Reticulocyte Responses in the Pigeon Produced by Material Effective and Noneffective in Pernicious Anemia with Description of the Histologically Different Reactions of the Bone Marrow." Page 1221. Address: Rutland State Sanatorium, Rutland, Mass.

Saklad, Meyer. M.D. Tufts College Medical School 1924. Anesthetist, Memorial Hospital, Pawtucket, Rhode Island; Rhode Island Hospital, Miriam Hospital, Providence Lying-In Hospital, St. Joseph Hospital, and Charles V. Chapin Hospital, Providence, Rhode Island. His subject is "Spinal Anesthesia. Agents, Methods and Indications." Page 1226. Address: 41 Angell Street, Providence, Rhode Island.

Rapoport, Boris. M.D. Tufts College Medical School 1914. Anesthetist, Beth Israel Hospital, Boston. Assistant in Surgery, Tufts College Medical School. His subject is "A Comparison of Postoperative Complications Following General and Spinal Anesthesia." Page 1235. Address: 270 Commonwealth Avenue, Boston.

Loman, Julius. M.D. Tufts College Medical School 1925. Research Associate in Psychiatry, Boston State Hospital. Instructor in Neurology, Tufts College Medical School. His subject is "Progress in Neurology in 1934." Page 1238. Address: 353 Commonwealth Avenue, Boston.

The Massachusetts Medical Society

A MEETING OF THE OFFICERS OF THE SCIENTIFIC SECTION

On the invitation of Dr. W. R. Morrison, Chairman of the Committee of Arrangements, the officers of the Scientific Sections of the Massachusetts Medical Society attended a luncheon at the Hotel Puritan, Boston, December 12, 1935 for the purpose of discussing the programs for the Annual Meeting of the Massachusetts Medical Society next June.

Dr. Charles E. Mongan, Dr. Channing Frothingham and Dr. Alexander Begg, officers of the Society, were also present.

Dr. Morrison reported the progress made by his committee in preparing the details of the facilities for the section meetings and the social features of the occasion, and called upon the chairmen of the seven sections for a report of plans under way. It was shown that all of the section officers have their respective duties well in hand and there is every assurance that the 1936 meeting will be of especial value to the members and their guests.

The President, Vice-President and Secretary submitted important suggestions for general consideration.

Dr. Morrison urged this representative body to pay especial attention to getting the recently elected members of the Society to attend and

become acquainted with the officers and familiar with the functions of the organization.

It was evident that everyone present was committed to making this meeting one of the best in the history of the Society.

ANNUAL MEETING

For the next annual meeting to be held in Springfield, Massachusetts, June 8, 9, and 10, the following subcommittees have been appointed:

Publicity Committee: R. S. Mace

Ladies Committee: W. A. R. Chapin

Historical Committee: G. L. Schadt

Scientific Committee: Frederick Jones

Hobby Exhibit Committee: E. P. Bagg, Jr.

Committee on Clinics: F. L. Dutton, Springfield Hospital; George B. Corcoran, Mercy Hospital; Frederick Hagler, Wesson Hospital

Sports Committee: Richard A. Rochford

SECTION OF OBSTETRICS AND GYNECOLOGY*

C. J. Kickham, M.D., *Chairman*, 524 Commonwealth Ave., Boston, Mass.

R. S. Titus, M.D., *Secretary*, 472 Commonwealth Ave., Boston, Mass.

Diagnosis of Sterility

Although the diagnosis of the causes of sterility is a difficult and time-consuming procedure when one is dealing with actual cases, it can be discussed quite simply.

There are four methods of diagnosis which are essential to a complete consideration of any case. The first is the number and character of the sperm cells. This can only be determined accurately by examination of the semen. Such an examination should estimate the number and vitality of the spermatozoa and should examine the individual cells for any morphologic changes. It should also determine whether the seminal fluid in which the spermatozoa are to be found is normally constituted.

The second test is made of the cervical secretions postcoitum. By this procedure it can be determined whether the sperm cells are able to get through the internal os in sufficient numbers so that they are practically certain to be able to reach the tubes. The test also shows whether the secretion itself is a favorable medium for this migration of the spermatozoa. Pus may be present or the mucus may be thick and stringy.

The third test is insufflation of the tubes. If they are normally patent no more need be done; if not, some material like lipiodol which is opaque to the x-rays should be injected and pictures taken.

The fourth procedure is to determine whether ovulation is occurring. This is best accomplished by getting an endometrial biopsy in the middle of the cycle and again at the end. From a study of stained sections of the endometrium one can postulate the occurrence of ovulation and tell a good deal about the hormone activity of the ovaries. Examination of the endometrium will also rule out endometritis. If all of these tests are satisfactory, one can predict pregnancy as being bound to occur sooner or later. If one or more of these tests are negative, treatment must be directed toward the correction of the conditions found before there can be any great expectation of success.

*A series of short selected articles by members of the Section is being published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

BOSTON MEDICAL LIBRARY

William Stokes, 1804-1878

As an illustration of the influence of heredity upon the development of exceptional intellectual qualities the life of William Stokes, a celebrated Irish physician of the early part of the nineteenth century, may be cited as especially noteworthy. Whitley Stokes, 1763-1845, his father, was the son of Gabriel Stokes, D.D., a fellow of Trinity College, Dublin. He was also prebendary of Elphin, Chancellor of Waterford and rector of Desart Martin. The first of the family to settle in Ireland was another Gabriel Stokes, great-grandfather of the subject of this sketch, who was an engineer and Surveyor General of Ireland in 1735. Whitley, the father of William, entered Trinity College in 1779, became a fellow in 1788, procuring his degrees of Bachelor of Medicine in 1789 and Doctor of Medicine in 1793. On account of an early affiliation with the United Irishmen, he came under the ban of English authority and was restrained from teaching for three years. However, he succeeded in removing the ban and was made a senior fellow in 1805 and a lecturer on Natural History in 1816. During 1830, he became the Regius Professor of Medicine, a position he held for thirteen years. So outstanding a figure did he become in the educational field that he was declared to be the best equipped man in the country to head a system of National Education in case Ireland should gain her independence of England.

Such was the immediate intellectual background of William Stokes who was born in Dublin in 1804, one of the nine children of Mary (Picknell) and Whitley Stokes, who were married in 1782. His education was conducted in the classics and mathematics by John Walker, a fellow of Trinity, and in the Sciences by his father. He pursued his medical studies at Edinburgh where he graduated in 1825. Re-

turning to Ireland that same year, he became a licentiate of the College of Physicians and was elected physician to the Meath Hospital, where he became a colleague of Dr. Robert James Graves. Pursuant to substantial work which he had evidently done while at Edinburgh, under Cullen, in pathology and clinical medicine, he published in 1825, a monograph on "The Use of the Stethoscope" which was the first article on this subject to appear in English. This he dedicated to Cullen.

From his opportunities at Meath Hospital and his most congenial association there with Robert Graves, with whom he collaborated in many ways, he became an inspiring teacher. In fact, one of his greatest contributions to medical interests has been the prominence that he gave to bedside teaching. Typhus fever visited Dublin in 1826 and Stokes was prominent through it all in his efforts to check its ravages. He married in 1828 and with an increasing practice, he moved to a more commodious home on York Street where he kept open house on Saturday evenings to which were attracted the cultured people of the town, for he had many interests outside his profession among which were art and archaeology. He was fond of collecting Irish antiquities and was the means of encouraging many a young man in various lines of artistic and scientific endeavor, notably George Petrie, the Archaeologist. He did not neglect any opportunities of foreign travel, which was of great assistance to him in developing his native, artistic tastes. These interests made him a great believer in the value of broad, cultural training to a physician and he frequently emphasized this point of view in his writings and teaching.

His hospital work, preparation of his teaching courses and the exactions of a large practice did not prevent him from doing a considerable amount of literary work, for in addition to his first work on the "Use of the Stethoscope" he brought out very valuable and extensive works on "Diseases of the Chest," "Diseases of the Heart and Aorta," "Clinical Observations on the Use of Opium," and numerous lectures and papers published in the London Cyclopaedia of Practical Medicine and elsewhere, covering a wide range of subjects. He founded the Dublin Pathological Society in 1838 and was for some years Editor of the *Dublin Medical Journal*.

To the profession in America the work done at Meath Hospital during the period of activity of Graves and Stokes should be of particular interest for it was largely from here that knowledge of the methods of bedside teaching, practiced by them, was brought back to this country. Though Boerhaave, one hundred years before, had introduced this plan of teaching, it was to the credit of these two that the method was widely introduced and popularized. This was doubtless due, so far as America was concerned, to the fact that fewer American students visited Leyden than came to Great Britain, and after the Edinburgh schools' influence began to wane, the school at Dublin enjoyed a short period (twenty years) of unusual popularity, largely due to the presence, there, of Graves and Stokes. During this time, many American youths found their way to Ireland and came under the spell of the teaching of these two master clinicians. It is a surprise to us, in this day, to realize what a battle had to be fought by those who were venturesome enough to employ the methods of Laennec and Auenbrugger in the study of diseases of the heart and lungs. It was due, in no small degree, to the championship of these methods that the renown of the Dublin School was achieved, as it was, of course, through their practical application that the discoveries in these fields, of such men as Graves, Stokes, Cheyne, Corrigan and Adams were made possible. It has been claimed by those competent to hold an opinion, that Stokes, after Corvisart, was the second greatest cardiologist the world has known.

Not only did these men cultivate the methods of clinical observation at the bedside but like all great diagnosticians and teachers they sensed the equally great importance of a knowledge of pathological anatomy, and the Dublin School became known in America as a place where such studies were prosecuted.

In 1861 he was made Physician-in-Ordinary to the Queen, in Ireland, and the same year was elected F.R.S. In 1874 he was chosen President of the Royal Irish Academy and in 1876 the Prussian Government awarded him the Prussian Order, Pour le Mérite, in recognition of his medical writings. This brief account of the life of a master mind may help us to see something more of a personality known to us chiefly, if not solely, because it bore a name associated with a peculiar type of respiration and a rare cardiac lesion.

MISCELLANY

THE APPOINTMENT OF DR. KARL BOWMAN

After a civil service examination of sixteen applicants, Dr. Karl M. Bowman, chief medical officer of the Boston Psychiatric Hospital, was selected to fill the position of Director of the Psychiatric Division of Bellevue Hospital.

DR. W. E. BROWNE ADDRESSES THE BRIDGEPORT MEDICAL ASSOCIATION

Dr. William E. Browne, Visiting Surgeon at Carney Hospital, Boston, recently was the guest speaker of the Bridgeport Medical Association. His exhibit of a variety of ingenious splints and the presentation of related cases made his address, "Management of Infections of the Hand," most interesting and instructive.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR OCTOBER, 1935

Disease	Oct., 1935	Oct., 1934	5 Yr. Average*
Anterior Poliomyelitis	237	10	88
Chickenpox	351	374	338
Diphtheria	35	70	157
Dog Bite	804	701	479
Epidemic Cerebrospinal Meningitis	13	5	6
German Measles	39	45	32
Gonorrhea	595	626	631
Lobar Pneumonia	259	181	192
Measles	179	91	212
Mumps	333	98	174
Scarlet Fever	552	479	567
Syphilis	511	404	373
Tuberculosis, Pulmonary	324	319	313
Tuberculosis, Other Forms	44	39	39
Typhoid Fever	13	15	27
Undulant Fever	8	2	
Whooping Cough	286	411	396

*Based on the figures for the preceding 5 years.

Rare Diseases

Anterior poliomyelitis—237 cases.

Dysentery (amebic) was reported from Saugus, 1.

Dysentery (bacillary) was reported from Worcester, 2.

Encephalitis lethargica was reported from Marlboro, 1; Peabody, 1; total, 2.

Epidemic cerebrospinal meningitis was reported from Boston, 2; Cambridge, 2; Deerfield, 1; Franklin, 1; Lawrence, 1; Longmeadow, 1; Lynn, 1; Medford 1; Somerville, 3; total, 13.

Malaria was reported from Andover, 1; Boston, 2; Chelsea, 1; total, 4.

Septic sore throat was reported from Arlington, 1; Beverly, 1; Boston, 4; Greenfield, 1; Newton, 1; total, 8.

Tetanus was reported from Medford, 1; Springfield, 1; total, 2.

Trachoma was reported from Boston, 3; Springfield, 1; Worcester, 1; total, 5.

Trichinosis was reported from Boston, 2; Methuen, 1; total, 3.

Undulant fever was reported from Millbury, 1; Natick, 1; North Adams, 2; Northampton, 1; Northbridge, 1; Upton, 1; Webster, 1; total, 8.

Résumé for October, 1935

Infantile paralysis for the State as a whole continued on the decline although a few communities noted a slight increase.

The decline of diphtheria continues to be striking. For the first nine months of the year there were sixteen deaths as compared with thirty-four in the corresponding period last year.

Tuberculosis pulmonary and other forms were reported about on a par with 1934.

Lobar pneumonia continues to run ahead of last year's figure and the indication is that it will continue to do so through the coming winter.

Scarlet fever was reported above last year's October figure and the indication is that the incidence this winter will be somewhat higher than in 1934.

Epidemic cerebrospinal meningitis is running at a definitely higher level than in 1934 with nearly every part of the State represented.

Typhoid fever was reported somewhat lower than last year. The number of deaths for the year to date (nine) is on a par with last year's figure, which was so low that some doubt was raised that it could be sustained.

Undulant fever was reported thirty-four times this year as compared with twelve for the year before, the increase being due to better reporting.

Although the number of reported dog bites is high, there were but nine reported cases of animal rabies as compared with twenty-seven in 1934.

Chickenpox and German measles are not remarkable.

Measles was reported somewhat higher than in 1934 but below the five-year average.

Mumps had its highest reported October incidence in the history of the State, while whooping cough was reported to a very low level.

THE NEW PHARMACOPOEIA

The U.S.P. Board Announces the Release Date and the Official Date for the U.S.P. XI

The new Pharmacopoeia, the Eleventh Revision, was available on December 16. This date was fixed by the Board of Trustees of the United States Pharmacopoeial Convention to enable the publishers, the Mack Printing Company of Easton, Pennsylvania, to place the books on sale in all parts of the country simultaneously.

As directed by the U.S.P. Convention, the Board of Trustees has also fixed the date when the standards of the new Pharmacopoeia shall become official, superseding the Tenth Revision. This date is June 1, 1936.

The following articles have been added to the Pharmacopoeia:

Acriflavina
Acriflavinae Hydrochloridum
Aethylenum
Aethylhydrocupreinae Hydrochloridum
Aethylis Oxidum (Solvent Ether)
Antitoxinum Scarlatinae Streptococcicum
Bismuthi et Potassii Tartras
Calcii Creosotas
Calcii Gluconas
Calcii Hydroxidum
Carbo Activatus
Carbonei Dioxidum
Chlorobutanol
Digitalis Pulverata (Biologically standardized)

Emulsum Petrolati Liquidi (50 per cent)
Ephedrina
Ephedrinae Hydrochloridum
Ephedrinae Sulfas
Erythritylis Tetranitras Dilutus (Erythrol Tetranitrate)
Extractum Hepatis
Ferri et Ammonii Citrates Virides
Fluoresceinum Solubile
Histaminae Phosphas
Hydrargyri Succinimidum
Iodophthaleinum Solubile
Liquor Ergosterolis Irradiati (Viosterol)
Liquor Hepatis
Liquor Hepatis Purificatus
Liquor Histaminae Phosphatis
Liquor Parathyroidei
Liquor Sodii Hypochloritis (4 per cent)
Merbaphenum
Neocinchophenum
Oleum Iodatum
Oleum Maydis
Oleum Morrhuae Non-destearinatum
Oleum Rosae
Phenacainae Hydrochloridum
Phenobarbitalum Solubile
Pulvis Chiniofoni
Serum Antimeningococcicum
Serum Antipneumococcicum—I
Sodii Perboras
Sodii Stearas
Stomachus
Tabellae Glycerylis Trinitratis
Theophyllina cum Ethylenediamina
Theophyllina cum Sodii Acetate
Tinctura Iodi Mitis (2 per cent)
Toxinum Diphthericum Detoxicatum
Toxinum Diphthericum Diagnosticum
Toxinum Scarlatinae Streptococcicum
Toxitabellae Hydrargyri Bichloridi Parvae
Tryparsamidum
Tuberculinum Pristinum
Vaccinum Rabies
Vaccinum Typhosum
Vaccinum Typho-paratyphosum

JAMES H. BEAL, *Chairman*,
WILLIAM B. DAY, *Secretary*.

PRESENT CONCEPTS OF TUBERCULOUS INFECTION

BY WADE HAMPTON FROST, M.D.
Professor of Epidemiology, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Md.

The most conspicuous fact in the history of tuberculosis in the last 50 years has been its steady and rapid decline in mortality. In view of the circumstances under which this has taken place and in the light of what is known of the natural history of the disease, is it reasonable to expect that the downward trend may continue indefinitely, perhaps to the point of regional suppression of the disease, or is it more reasonable to anticipate a compensating upward swing to a higher level?

The fact that the tubercle bacillus is an obligate parasite of man, transmitted directly from person to person by way of the respiratory tract, places tuberculosis in the group of diseases offering least hope of full control except by specific immunization. However, tuberculosis is sharply differentiated from the other diseases in this group by the fact that two restrictions are imposed upon the propagation of the tubercle bacillus, namely, (1) in order to spread the infection, it must produce actual disease—a lesion which becomes "open"; and (2) it succeeds in producing this effect in only a limited proportion of those who become infected. Various other pathogenic organisms transmitted by way of the respiratory tract are subject to one or the other of these restrictions, or its equivalent, but the tubercle bacillus is unique in being subject to both.

Because of this combination of conditions limiting the propagation of the tubercle bacillus, it is reasonable to infer that the *partial* environmental control which has been established is definitely effective in limiting the spread of tuberculosis even though it be relatively ineffective against numerous other diseases transmitted directly by way of the respiratory tract. The fact that at present tuberculous infection, as distinguished from the disease, is highly prevalent, is not inconsistent with this view.

With improved measures of control which are within the limits of practicability, including better detection and isolation of open cases, with higher standards of living and personal hygiene, there appears to be no fundamental reason why tuberculosis may not be eradicated eventually from large areas in this country. There are, indeed, certain contingencies which obviously might bring about a recrudescence after the disease has reached an extremely low level, but it does not appear that this result is inevitable in accordance with any accepted biological law or that it is especially to be anticipated.

Admitting that we cannot actually know the future of tuberculosis, it is none the less important to define clearly what are reasonable expectations in the light of present knowledge, since present activities in study and control necessarily are directed chiefly toward the future. If, as now appears, it is reasonable to anticipate eventual control to the point of permanent regional suppression, the establishment of this as the objective has obvious and important implications as to the scope and intensity of control measures. It has less obvious but important implications with respect to indicated lines of investigation.

Through the courtesy of the N. T. A. and of Dr. Frost, the above was published in *Neighborhood Health*, August-September, 1935, Vol. 1, No. 2.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1934 AND SEVEN YEAR AVERAGE

MONTH ENDING DECEMBER 7, 1935

Diseases	1935 Week ending Nov. 16	1935 Week ending Nov. 23	1935 Week ending Nov. 30	1935 Week ending Dec. 7	Average cases reported for week corresponding to Dec. 7 for past seven years	1934 Week ending Nov. 17	1934 Week ending Nov. 24	1934 Week ending Dec. 1	1934 Week ending Dec. 8
Chicken Pox	96	121	89	149	147	73	199	142	291
Diphtheria	3	1	4	9	14	3	1	2	3
Dysentery Bacillary	1	—	—	—	—	4	1	6	—
Encephalitis Epidemic	1	—	—	—	—	1	—	—	—
German Measles	9	13	19	4	6	1	—	—	16
Influenza	3	2	18	9	5	4	1	1	1
Measles	52	55	29	110	75	209	222	258	272
Meningococcus Meningitis	1	1	2	—	—	—	—	2	—
Mumps	29	63	45	65	48	20	13	16	24
Paratyphoid Fever	—	2	1	—	—	—	1	—	—
Pneumonia (Broncho)	19	9	12	24	30	16	24	15	21
Pneumonia (Lobar)	17	18	13	34	32	16	17	16	17
Poliomyelitis	3	6	5	2	—	—	—	—	—
Scarlet Fever	27	54	32	33	58	34	45	38	36
Smallpox	—	—	—	—	7	—	—	—	—
Streptococcus Sore Throat	2	1	—	1	1	—	6	1	4
Trichinosis	2	—	—	2	—	4	—	4	3
Tuberculosis (Pul.)	23	22	13	26	23	11	44	31	15
Tuberculosis (O. F.)	2	1	1	3	2	1	1	1	2
Typhoid Fever	2	1	—	1	3	—	2	—	1
Undulant Fever	3	2	4	3	1	—	1	1	4
Whooping Cough	82	110	75	71	63	72	65	36	89
Gonorrhea	27	34	26	22	51	23	30	41	54
Syphilis	26	42	37	49	58	30	64	67	51

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

"MAKING WAR AND MAKING PEACE"

That psychiatry, which played an important part in preparing men to be soldiers in the World War has an even greater responsibility in preparing them for peace was the keynote of an address delivered by Dr. Stewart Paton at the twenty-sixth annual meeting of The National Committee for Mental Hygiene held at the Rockefeller Center, New York City, November 14, 1935. Dr. Paton, formerly lecturer on neurobiology at Princeton and other universities, spoke on "Making War and Making Peace."

"One of the lessons that we learned during the war," Dr. Paton said, "was that in order to insure military success it was necessary for the soldiers in active service to be sound in mind and body. The late Dr. Thomas W. Salmon, first Medical Director of The National Committee for Mental Hygiene, and his associates, did a great deal to open the eyes of military leaders to the importance of the emotional and mental preparedness that is necessary in order to make a good soldier. But as we have been exceedingly indifferent and reluctant about informing ourselves as to the emotional and mental preparedness required to be peaceful and human, relatively little progress has been made in finding ways and means for the pacific settlement of either individual or international problems."

Dr. Paton expressed the opinion that the National Committee's outstanding contribution during the past quarter of a century has been in bringing about public realization that our most valuable possessions are our distinctive human characteristics, and that this must become an essential part of our outlook if we are to hope for an end of international strife.

"Man is gradually learning to fuse impulse and reason, and unless the strain is too great, he often succeeds in being peaceful, sane and human," he said. "However, the problem of how to make peace is a much more difficult one than how to make war,

and as we have given more intelligent consideration to the war than to the peace problem, we should not be surprised nor shocked by the difficulties we experience in settling any of our conflicts."

Dr. Paton pointed out that while there is universal agreement that our military leaders may be physically and mentally sound, we pay little or no attention to the equally important requirement of sound mental and emotional qualifications in our governmental and political leaders who are engaged in the vital business of diplomatic negotiation, discussion and arbitration. A great deal of valuable energy is wasted in promoting rather than in practicing peace, he said, due to the lack of appreciation that the rational and peaceful settlement of disputes requires definite and particular techniques and method of approach.

"Too many people are now engaged in trying to promote peace throughout the world who would not be competent to assist an individual in finding a peaceful solution of the problems that drive him to take refuge in a mental hospital."

Here Dr. Paton referred to the warning recently issued by the Netherlands Medical Association, in a statement signed by 339 psychiatrists from thirty nations, which pointed out the war dangers of the present world situation, and which, he said, suggested the constructive psychological methods that should be adopted to avoid destructive and insane conflicts.

"Although we have learned from hard and trying experience that men cannot be prepared for constructive service if they adopt prohibitive attitudes of mind," he said, "we still rely, to a large extent, on prohibitions to make us temperate and sane. As a result, mental hospitals and clinics are overcrowded with patients who have not been able to settle the conflicts that disturb their lives on a rational and peaceful basis."

One of the basic factors that must be considered in planning for world peace, Dr. Paton further said, is our "emotional immaturity".

"One of the signs that we are still children, psychologically speaking, is the intense interest we take in all the sensational aspects of life," he said. "For centuries we have focussed attention upon going to war, going insane, going unemployed, and going to the devil. Very little notice, however, is taken of the process of going more human, more peaceful, more sane. Yet, slowly but surely, we are becoming aware of the fact that man is a human being, in spite of the fact that the present plague of nervous and mental disorders, the greatest epidemic that has ever afflicted mankind, is seriously interfering with our attempts to be peaceful, sane and human.

"Mental hygiene, as time goes on, can assist the American people in finding peaceful and rational solutions of their personal, business, industrial and political problems. By continuing and supplementing the work that The National Committee for Mental Hygiene has already done, much can be accomplished in opening our eyes to the significance of the fact that we are human beings.

"One of the dangers at present is that technical science has supplied us with means for annihilating the human race. This catastrophe can be avoided by securing the active coöperation of science and art, and mental hygiene can show the way in which we can secure this coöperation that is so essential to progress in the art of living."

CARE FOR INDIGENT HEART CASES IN CONNECTICUT

Mrs. F. Donald Coster of Fairfield, Connecticut, has developed plans for the reëstablishment of a free clinic for indigent persons afflicted with heart diseases in Bridgeport, Connecticut. When the clinic is in full operation, fifteen trustees will be in charge of the endowment.

The facilities of the clinic will be available to any resident of Connecticut who is unable to pay for treatment.

CORRESPONDENCE

CONSULTATION *A LA MODE*

Editor, *New England Journal of Medicine*,

One of the handicaps of being a general practitioner is the painfully humiliating experience he is constantly called upon to go through at the hands of the well-meaning specialist who sees nothing unethical in the usual procedure of lifting all the responsibility from the shoulders of the doctor who calls him in *consultation* and thereafter giving said patient such excellent attention that the erstwhile physician—in good and regular standing with his former patient—is utterly ignored and forgotten.

In spite of all that has been said and written to the contrary, this baneful practice still goes on and it often seems true that the greater the man called in consultation the more inevitable the outcome. Today, calling a consultation with many of these men of otherwise high standing is tantamount, not only to giving up your patient, but your professional standing and prestige with all concerned. The process is apt to be about as follows:

Consultant suggests removal to hospital where case may be watched and studied. Very good. You would like to watch and study this patient yourself and that's why you called him in. Perhaps you simply wanted his judgment as to whether case is operatively urgent. From courtesy you accept the first suggestion. Presto! Your patient is removed *as a private case* to a semi-private institution with a "closed staff".

You make your first call full of interest and expectancy, only to find your patient well established under the more or less exclusive care of Dr. Dash—who "covers for Dr. Blank"—a man who doesn't know you from Adam and whom you did not ask for advice. You are told courteously that Dr. Blank said you were welcome to "come in" any time; meanwhile "we" are working up your case. Patient one day requests you to prescribe an aspirin for her headache and discovers it cannot be done.

You make several visits, you see your patient in

bed (under covers) but you are never asked advice about any procedure whatsoever, or expected to make an examination. Interest flags. The patient seems well satisfied and thanks you patronizingly for sending her to such a nice place, and quotes Dr. Dash whom you now rarely see, or Dr. Blank who "looks in occasionally". "Very big man, you know, did ten operations yesterday," Dr. Dash assisting.

Eventually you learn that Dr. A has been called to examine nose and throat; Dr. B the eyes, and Dr. C is "going over" the heart and kidneys preparatory to operation. Finally you are *invited* to "witness" the operation. You may have thought you were something of an authority on some one of these lines yourself, but no questions being asked, your self-confidence gradually weakens so that when you receive the final notice of the patient's discharge with "follow up nurse" you accept the inevitable. Patient to report monthly to Dr. Blank.

As a social duty you call at the house of your patient and are told everything is being taken care of and you will be sent for if needed. When the month comes around you debate on the policy of sending any bill whatsoever for services—except the initial call ($3.00). If wise, acting on past experience, you simply pass it up to profit and loss, and wonder who, among the great luminaries of modern medicine, will be your next beneficiary in the sum of some hundreds of dollars.

As a physician of thirty-five years' experience, and near the retiring age anyhow—there is little to say from the standpoint of personal grievance. I am merely pointing to one of the conditions which make general practice difficult and frequently humiliating in the extreme. The doctor today not only sees his poorer patients drifting to the clinics (often run by young physicians and admiring nurses who "make suggestions") but his richer patients constantly being beguiled by a process so subtle that it takes his breath away and leaves him wondering like the man in the play: "Am I now as big a fool as I think I am?"

What can be done about it? Unfortunately, the problem is not an easy one and it is futile to pass it up by implying unworthy motives to anyone. There was a time, within the memory of individuals now living, when it was reasonably safe for the family physician to call a first-class consultant without the common danger of being utterly supplanted or ignored in the further conduct of the case. His opinions were often sought and respected *in the presence of the patient or members of his family.* "Follow up" engagements were arranged by mutual consent. If the case proved to be one for hospital care the consultant and the general man were in constant sympathy and intimate touch with the patient.

Whether this relation of mutual respect and genuine coöperation can continue under modern conditions in such manner as to prevent the humiliating situations above outlined is difficult to say. On the whole I have nothing but profound respect for most of the talented surgeons and specialists who have assisted me over many difficult places in the past. On the surface it would appear that the general practitioner has no other recourse than to *refuse to have his private patients transferred to any closed staff hospital unless he himself is on that staff.* If there is any other way to avoid the inevitable situation above suggested, the writer, as a fair representative of the fast-disappearing fraternity of the general practitioner, would like to hear from those specialists in a position to speak for themselves.

WILLIAM W. HARVEY, M.D.
114 Fenway, Boston.

CONCERNING JENNER AND JOHN HUNTER

Editor, *New England Journal of Medicine,*

The following is from "The Farrington Diary" by John Farrington, R.A., edited by James Greig. George H. Doran and Company, 1923:

"'Of Vaccination Fame"

"September 13" (1796) "Dr. Jenner was some years ago with John Hunter (The famous surgeon), and had He preferred a town life might have been connected with him in business. . . . He knows Loutherburgh, and observed that He does not receive remarks on his work graciously.

"While Loutherburgh was painting one day John Hunter remarked that a certain part was *too green,* . . . 'Not green enough,' said Loutherburgh, and dipping his pencil in the strongest green colour put it on the canvass.

". . . Foote, the surgeon, became rancorous against John Hunter, because the latter had seemed to describe a Bougie which Foote had invented, as not necessary. . . . To revenge himself He wrote of Hunter with much malignancy and asserted many falsehoods. . . .

"Dr. Jenner has a great opinion of the Cheltenham Waters, . . . but they may be drunk imprudently which He sees in the countenances of many Young Ladies at the well. . . . Above 3000 people have drank them this Season, . . . not one who came for the benefit of them has died. . . .

"September 24th. Dr. Jenner has found that in *insane patients.* He has moderated their violence by keeping them sick with tartar emetic. He observed that a person is more liable to take cold who suddently removes from *cold* to *heat* than from *heat* to *cold.* Camphor water is an excellent medicine for nervous complaints.

"September 27th. . . . Dr. Jenner shewed us some lines which the Revd. Dr. Stevens gave him as having been written by Gray as part of his Elegy in a Country Church Yard, but were omitted. . . .

" 'Some rural Lais with all conquering charms,
'Perhaps now moulders in the grassy bourne,
'Some Helen, vein to set the fields in arms,
'Some Emma dead of gentle love forlorn.' "

Very truly yours,

WM. PEARCE COUES, M D.
12 Monmouth Court, Brookline, Mass.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Illinois,
December 5, 1935.

Editor, *New England Journal of Medicine*,

In addition to the articles enumerated in our letter of November 2, the following have been accepted:

The Calco Chemical Co., Inc.
Tablets Methenamine—Calco, 5 grains

Cutter Laboratories
Diphtheria Toxin for the Schick Test, Diluted Ready for Use
Diphtheria Toxin-Antitoxin Mixture 0.1 L+ (Goat) ten 3 cc. vials package
Typhoid-Paratyphoid Prophylactic, 10 vials package

Lederle Laboratories, Inc.
Gas Gangrene Antitoxin (Polyvalent) Without Tetanus Antitoxin, "Globulin-Lederle-Modified"
Tetanus Gas Gangrene Antitoxin, "Globulin-Lederle-Modified"

Parke, Davis & Co.
Gas-Gangrene Antitoxin (Combined) Refined and Concentrated—P. D. & Co.

Yours sincerely,
PAUL NICHOLAS LEECH, *Secretary*.

RECENT DEATHS

CAHILL—CHARLES SUMNER CAHILL, M.D., of 311 Prospect Street, Cambridge, Massachusetts, died at his home, December 10, 1935, aged seventy-one years. He graduated from the Harvard Medical School in 1886 and was a Fellow of the Massachusetts Medical Society and the American Medical Association.

Dr. Cahill was active in the formation of the Cambridge City Hospital and served as chairman of the board of trustees from 1912 until December 1931. He was active in civic affairs having served on the park commission of Cambridge and as an official of the Inman Coöperative Bank and the Central Trust Company. He was a member of the Cambridge Lodge of Elks and the Cambridge Knights of Columbus.

His brother, Dr. Thomas J. Cahill, of Cambridge, and four sisters survive him.

LAWLOR—EDWARD FRANCIS LAWLOR, M.D., of 33 Tremont Street, Lawrence, Massachusetts, died at his home, October 6, 1935. He was born in Lawrence in 1877, educated in the public schools of that city and graduated from the Baltimore Medical College in 1905. He began practice in Lawrence soon after graduating in medicine and joined the Massachusetts Medical Society and the American Medical Association.

For a year and a half he suffered with organic diseases.

He was the surviving one of three brothers who practiced in Lawrence. He is survived by his widow, Mrs. Florence Lawlor; a daughter, Elizabeth Lawlor; a son, Dr. Edward F. Lawlor, an interne at the Salem Hospital, and several sisters and brothers.

LOVITT—ISRAEL MELBOURNE LOVITT, M.D., of Yarmouth, Nova Scotia, died at his home, December 7, 1935.

He graduated from the Harvard University Medical School in 1885, then spent a year in European hospitals.

He began medical practice in Yarmouth but soon discontinued it to engage in business and philanthropy.

Several children survive him.

NOTICES

AMERICAN BOARD OF OPHTHALMOLOGY

The 1936 Examinations will be held in Kansas City, May 11, at the time of the American Medical Association meeting, and in New York City, in October, at the time of the American Academy meeting.

All applications and case reports must be filed at least sixty days before date of examination. For information, syllabuses and application forms, address Dr. Thomas D. Allen, Assistant Secretary, 122 South Michigan Avenue, Chicago, Illinois.

ANNOUNCEMENTS

CHARLES E. DUMAS, M[illegible] announces the opening of his office at 390 Main [illegible]orcester.

JOSEPH HAHN, M.D., [illegible]es the opening of an office at 146 Chestnut St[illegible] Springfield, Mass.

REPORTS AND NO[illegible]ICES OF MEETINGS

THE WILLIAM HARVEY SOCIETY

The William Harvey Society met November 8, at the Beth Israel Hospital, Dr. Cadis Phipps presiding. Dr. E. B. Pardee, Professor of Medicine at Cornell Medical School, spoke on "Arteriosclerotic Heart Disease". Dr. Pardee emphasized the fact that generalized arteriosclerosis is not an indication of heart disease unless there is also sclerosis of the coronary arteries. Atherosclerotic changes in the cardiac valves may lead to aortic insufficiency or to mitral stenosis, but such changes are usually slight, unimportant pathologically, difficult to diagnose, and are rarely the primary cause of cardiac symptoms, although they may contribute to such symptoms as are caused by other disease processes.

Hypertension is a frequent complication of arterio-

sclerotic heart disease, although its exact rôle is difficult to state. Dr. Pardee believes that persistent hypertension can give rise to arteriosclerosis, both generalized and coronary. On the other hand hypertension can cause cardiac hypertrophy and failure without the presence of any coronary disease whatsoever.

There are three forms of coronary arteriosclerosis:

(1) Generalized narrowing of all the coronary system, with degeneration of the muscle cells and fibrous tissue replacement.

(2) Slow narrowing of one or more branches of the coronary system with resultant localized fibrotic changes. There is usually an associated generalized process of less severity. It is in this type that slow but complete limited occlusion may occur without clinical manifestations.

(3) Abrupt thrombosis of a previously patent vessel with myocardial degeneration and subsequent rupture or fibrosis. If the thrombosed vessel is small, and if collateral circulation is good, the involved area may escape acute degeneration.

One of these forms is usually predominant, but the other processes are usually found in greater or less degree in the same heart.

Although arteriosclerosis is the most usual cause of myocardial fibrosis, it is also a result of chronic passive congestion, chronic rheumatic involvement, or of narrowing of the coronary mouths due to luetic aortitis or rheumatic aortic disease with calcification.

Coronary disease is usually manifest by some or all of the following characteristic symptoms:

(1) The angina of effort which subsides with the cessation of exertion. Occasionally there is recurrence of pain at night, which is relieved when the patient assumes a sitting position. This type of pain is most often associated with localized narrowing of a coronary branch.

(2) Severe prostration and persistent angina occurring with coronary thrombosis. The prostration may persist after the disappearance of the angina, or may be the only manifestation of coronary occlusion.

(3) Chronic cardiac insufficiency, characterized by dyspnea on exertion which steadily progresses in severity over a period of years. Eventually shortness of breath is existent while the patient is at rest. This dyspnea is usually accompanied by some degree of cough. These patients may complain primarily of easy fatigue, rather than dyspnea. Ankle edema and hemoptysis are relatively late symptoms. Nocturnal dyspnea of paroxysmal or Cheyne-Stokes type is often a troublesome manifestation.

(4) Cardiac arrhythmias are commonly encountered. Of these premature beats are the most common. Auricular fibrillation is of frequent occurrence; if paroxysmal, causing distress at the moment of change of rhythm, and if persistent, attracting attention only after exertion or excitement. Heart block is relatively rare, resulting in Adams-Stokes syndrome in its advanced form.

Just as the pathological features are usually mixed, so are the symptoms. However, certain correlations between the pathological and clinical manifestations of the disease may be summarized as follows:

Predominant Pathology	Usually Related Symptoms
A. Generalized coronary narrowing.	A. (1) Cardiac insufficiency. (2) Cardiac arrhythmias.
B. Localized coronary narrowing.	B. (1) Angina of effort. (2) Paroxysmal nocturnal dyspnea. (3) Cardiac arrhythmias.
C. Acute coronary thrombosis.	C. (1) Acute persistent angina and prostration. (2) Cardiac insufficiency. (3) Cardiac arrhythmias.

Analysis of one hundred ambulatory patients with arteriosclerotic heart disease revealed the following incidence of initial symptoms:

Angina of effort	33%
Shortness of breath	24%
Coronary thrombosis	34%
Palpitation	5%
No symptoms	4%

The incidence of coronary thrombosis as an initial symptom is undoubtedly greater than the 34 per cent noted above, inasmuch as many patients do not survive the first attack.

Those patients with coronary thrombosis as the initial manifestation of heart disease have a much higher incidence of subsequent thromboses than do patients with initial symptoms of cardiac insufficiency. The time between subsequent thromboses is extremely variant, being as long as twenty years in some cases. Giving a prognosis as to life expectancy in such cases on the basis of average time between attacks is useless, and is to be condemned inasmuch as it may cause undue alarm on the part of the patient's family.

The diagnosis of arteriosclerotic heart disease is confirmed by the presence of certain physical signs. Such signs are derived from four main sources:

(1) Inasmuch as atheroma of the aorta is usually accompanied by coronary arteriosclerosis, signs indicative of the existence of aortic atheroma are to be considered as evidence for the co-existence of coronary sclerosis. Thus, the presence of a hard-ringing aortic second sound (in the absence of hypertension), a harsh systolic murmur at the aortic area, and x-ray findings of aortic atheroma and prominent aortic knob, are strong evidence for the existence of coronary arteriosclerosis.

(2) Cardiac enlargement in the absence of hyper-

tension or valvular disease is indicative of arteriosclerotic heart disease.

(3) A faint first heart sound with a normal or increased sound shows myocardial weakness and points to coronary sclerosis. Prolongation or duplication of the first sound and gallop rhythms are of similar importance.

(4) Electrocardiographic changes when properly evaluated are of distinct value in differentiating various cardiac lesions. Electrocardiographic abnormalities in patients of a given age without other possible causes are to be considered as due to coronary arteriosclerosis. The most important of these suggestive changes are the following: inversion of the T waves in the first or second leads, a diminished amplitude of the P wave, a small or biphasic T wave, a prolonged P-R interval, a small amplitude of the Q-R-S complex, or a low take-off of the T wave.

BOSTON ORTHOPEDIC CLUB

The second meeting of the Boston Orthopedic Club for the year was held in the Boston Medical Library on November 12, 1935. Dr. George W. Van Gorder opened the meeting and briefly introduced the speaker of the evening, Dr. Sumner M. Roberts. Dr. Roberts spoke on "Dislocations and Fractures of the Cervical Vertebrae." Contrary to the opinion commonly held, a "broken neck" does not always mean death and if treated properly, the majority of cases recover. As in the treatment of all fractures, accurate knowledge of the regional anatomy is essential, and Dr. Roberts discussed the structural differences between the cervical region and the rest of the spine. The range of motion is distinctly greater, particularly in the upper part of the cervical spine, than in the lower vertebrae, and because of the location, this region is especially susceptible to injury. Accidents resulting in damage to this region usually consist in a forcible forward flexion of the neck.

Stiffness of the neck may be the only sign of a slight fracture. Tenderness over the transverse and spinous processes of the involved vertebrae is usually present and may be very marked. There is frequently a definite abnormality in the position of the head. If the fracture is very severe, there may be pain and sensory disturbances in the arms as well as paralysis. This may be due to hemorrhage or edema of the cord or surrounding tissues and may be transient. If there is no evidence of cord damage, and this should be carefully ruled out, the patient may sit up to be examined. When the head is held in both hands by the examiner and gently lifted upward, the pain is frequently relieved. The neck should not be flexed forward during the examination. X-ray examination is often necessary to make a definite diagnosis. Dr. Roberts has found lateral "stereo" views to be useful.

Fractures or dislocations of the cervical spine must be reduced just as any other similar injury elsewhere, and the sooner this is done, the better the results will be. Closed methods should be used except on rare occasions. Preliminary to reduction, the patient is put flat in bed on a Bradford frame with a pillow under the shoulders and the head in a simple canvas sling and five pounds of traction exerted. The head may be gradually hyperextended by the use of a collapsed pillow, under the shoulders, which is slowly inflated with an air pump. After reduction has been accomplished, a plaster cast is applied. This cast should extend over the entire head with only a hole for the mouth, nose, ears and eyes and perhaps another for the topmost part of the head. The chin and forehead should be well padded with felt; and a window should be placed over the Adam's apple. After the cast, a molded leather collar is used with a band extending around and over the brow.

The simplest type of fracture is of the spinous process. These are often multiple and require four to six weeks of fixation. Dr. Roberts has had five cases of fracture of the odontoid process in his series. The fracture usually takes place at the base of the process and a resulting dislocation of the atlas is common. This fracture gives pain high up in the neck which spreads into the occiput and there is pain on rotation of the head. Careful x-rays through the mouth should be taken to establish the diagnosis. A period of two to three months of complete fixation in plaster is necessary and this must be followed by two months with a molded leather collar and then finally two more months with a Thomas collar. For the treatment of compression fractures, Dr. Roberts gradually hyperextends the neck over a period of forty-eight hours by means of the pneumatic pillow, at the same time exerting five to eight pounds' traction. Exercises can be begun gradually after two months. These fractures are more apt to be in the lower portion of the cervical spine. Fractures of the laminae, articular facets, and pedicles were discussed briefly.

Dr. Roberts has had nineteen dislocations in his series. They are most frequent in the upper portion of the cervical region where the articular facets are almost horizontal. They are often accompanied by small fractures and are usually caused by fairly severe injury. There is usually pain and tenderness at the site to help localize the lesion. If the dislocation is complete on one side, the head is tipped to the other side and the chin tilted to the same side. These conditions should be reduced immediately, by traction and hyperextension if incomplete, and by closed manual manipulation if complete. For the latter process complete anesthesia is necessary and forward flexion of the head must be prevented. The patient is placed on his back with his head flush with the end of the table and a strip going around the chin and around the doctor's waist. The head is turned to the same side as the dislocation, then tipped to the other side and brought back to the midline (Walton's maneuver). As the dislocated bone slips back into place, a click is often heard. Reduction of dislocations may be attempted as long as ten months after injury if there is a

definite deformity. If the dislocation is complete, there should be complete fixation for two months followed by two months each with the leather molded collar and with the Thomas collar. If the dislocation is incomplete, the last two procedures are all that is necessary.

The early complications, before reduction has taken place, consist in transient or permanent neurological disturbances in the arms. These may be caused by nerve root pressure, hematomyelia, or cord compression. Seventy-five per cent of the fracture cases showed cord symptoms while only ten per cent of the dislocations had this complication. The late complications arise from cord damage resulting from overgrowth of callous and scar tissue. These can best be prevented by early reduction and adequate fixation.

Operation is rarely necessary for reduction of fractures or dislocations in this region. Rarely is a stabilization procedure necessary. If a block is present as shown by lumbar puncture, and remains after reduction, laminectomy may be indicated. This operation has a high mortality.

The mortality for the whole series of thirty-seven cases was only nineteen per cent; twelve per cent for the dislocations; and forty-two per cent for the compression fractures.

In the discussion which followed, Dr. Greene discussed the case of a child with Still's disease who had a complete dislocation between the first and second cervical vertebrae which was probably due to an involvement of the bursae around the odontoid process.

THE GREATER BOSTON MEDICAL SOCIETY

The Greater Boston Medical Society met at the Beth Israel Hospital November 5, Dr. Harry Linenthal presiding. Dr. Harold Neuhof of the Mt. Sinai Hospital, New York, delivered an address on "The Pathogenesis, Pathology and Treatment of Putrid Abscess of the Lung, with Special Reference to the Surgical Treatment of Acute Abscess and Its Results."

Dr. Neuhof stressed the common occurrence of putrid abscess of the lung, and the fact that from the point of view of the patient it is of more importance than carcinoma of the lung. Untreated cases rarely live more than three years, the fatality of such cases being fully as high as that of carcinoma. Properly managed by surgical measures the fatalities are relatively few.

Dr. Neuhof considers that the only suppurative abscess of the lung which is of clinical importance is that caused by anaerobic organisms, and containing putrid pus. He differentiates this condition and nonputrid pulmonary suppuration of staphylococcus, streptococcus, or pneumococcus etiology. In his experience nonputrid abscess of the lung does not exist as a primary disease. Cases which seem at first to be such, always prove to have some other underlying pathological process, such for example as bronchiogenic carcinoma.

The pathological features of acute putrid abscess of the lung are quite characteristic. They are solitary, spherical in shape, unilocular, involve a bronchopulmonary segment, and are always of appreciable size. They are always near the periphery of the lung with never more than a thin shell of fibrotic, avascular lung tissue lying between them and the pleural cavity. Central pulmonary abscesses do not exist, although lesions lying near the interlobar septum or near the mediastinal pleura may appear centrally located in x-ray plates. There is pleural irritation over the region occupied by the involved bronchopulmonary segment with resultant formation of adhesions and obliteration of the pleural cavity. These adhesions are of sufficient strength within two or three weeks after onset to allow operative traversal of the area without entry of the pleural cavity. Putrid abscesses invariably communicate with a bronchus, and as a result there is practically always some foulness of the sputum, the breath, or of the secretions found at bronchoscopy.

A lung abscess as here defined is an abscess from its inception. Except in very rare instances such as pneumonitis following partial drowning or suffocation, there is no preceding pneumonitis. Neither is there a bronchial infection with transition to an abscess. By the time an abscess is seen clinically it is a localized pocket of pus with surrounding chronic and acute inflammation.

An arbitrary limit of six weeks has been set as the dividing line between acute and subacute or subchronic pulmonary abscesses. Although there is marked variation in individual cases as to the exact time when the symptoms become those of a subacute process instead of those of acute infection, six weeks is quite satisfactory for statistical separation of the two stages.

In the subacute or subchronic abscess, fibrosis has become fairly well established. Local spill-over of pus into adjacent lung segments is very probable. Surrounding bronchiectasis is prone to develop. An irreversible process is thus apt to be initiated, and once firm chronicity is established management and cure may become impossible.

The pathology of the chronic abscess is quite distinct from that of the acute disease. Fibrosis is marked, the abscess is multilocular and often multiple. Bronchiectasis develops, and may become so severe as to conceal the underlying abscess. The previously outstanding putridity becomes masked, and may fall into the background, the sputum showing only occasional foulness.

Rupture of the abscess into the pleural cavity with resultant empyema may occur at any stage of the disease, and although not of hopeless prognosis is a very serious complication. The concept that such an occurrence is to be regarded as a favorable relief of the abscess is entirely false.

Although in rare instances drainage of the empyema will result in healing of the abscess cavity, this is by no means the generally encountered feature. Most such cases require treatment of both the empyema and the abscess.

The pathogenesis of putrid lung abscess is still obscure, and a disputed subject. Dr. Neuhof believes that most cases are of aspirational origin. If they were of embolic origin the following features would be expected: The lesions should be multiple, and related to blood vessels. There is no reason why they should be related to the bronchial tree. They would be expected to develop at any place in the lung parenchyma, and would not always be peripheral in situation. Their size would be quite variant, and metastatic lesions should also be found in other organs. If the abscess is putrid there should be some feeding sources, also putrid. It is evident that this picture is quite different from that observed in the usual case suffering from putrid abscess.

Studies made at the Mount Sinai Hospital in New York have supported the aspirational theory of pathogenesis in finding a possible and quite probable source for the aspiration of infected material in the dental caries present in all cases in which no definite source for the abscess could be found.

The signs and symptoms of putrid abscess are fairly characteristic and should serve to differentiate this condition and pneumonia. Chest pain due to pleural reaction is never absent. It is a sharp, definitely localized pain, with frequent associated local tenderness. Cough is present, and usually not productive in the early stages. Hemoptysis is almost always present, being found even more frequently than in tuberculosis. Occasionally, death occurs from hemorrhage. Fetor is usually present, and may be either early or late in appearance. It varies from a slight odor to foul sputum, and is occasionally noted only on bronchoscopic examination. Metastatic abscesses to the brain occur in the chronic form of the disease most commonly.

The course of the disease varies from benign to fulminating. Twenty to thirty per cent recover spontaneously. In Dr. Neuhof's clinic the percentage of spontaneous cures has dropped to but ten per cent. This drop is probably explained by the fact that operative therapy is employed in many more cases than formerly. The subacute phase may develop very insidiously without the presence of foulness or other symptoms. For this reason apparent spontaneous cures of acute abscesses should be followed closely.

The physical signs are often absent and when present are not characteristic. They are of apparent insignificance in the face of the other manifestations of the disease.

X-ray studies are of great diagnostic importance. The presence of a fluid level surrounded by an area of increased density is pathognomonic, and confirms the diagnosis. Although such a lesion may appear to lie in any part of the lung field, it should be remembered that the apparently mesially situated abscesses are actually situated posteriorly. Anteroposterior and lateral views should be taken on all cases.

X-ray films showing a pneumonitis with a small fluid level usually represent a large abscess with a surrounding inflammatory reaction. A fluid level is always indicative of connection with a bronchus, and shows that partial drainage has occurred. The disappearance of the cavity without diminution of the infiltration is not a sign of cure. Films actually showing a centrally situated lesion rule out the diagnosis of lung abscess. The x-ray findings may simulate fibroid tuberculosis, and indeed abscess may be present in addition to this condition. Finally it should be remembered that it is often impossible to distinguish between cancer of the lung and putrid abscess roentgenologically.

Bronchoscopic examination is advised in all subacute and chronic cases and in the majority of acute cases. It is of great value in determining the exact location of the lesion and in establishing the presence of foreign body, neoplasm, or other etiological agent. Bronchoscopy when properly performed causes very little discomfort to the patient. The bronchoscope will not enter the abscess cavity unless undue force is used.

The treatment of putrid abscess of the lung should always be surgical. Attempts at drug therapy, postural drainage, and bronchoscopic irrigation have all proved futile. Pneumothorax is unreservedly condemned, since such therapy usually precipitates a fulminating and fatal empyema.

The majority of cases should be operated upon while still in the acute phase of their disease, preferably two or three weeks after the onset. Only cases presenting small lesions with a mild course should be given expectant therapy.

Dr. Neuhof reports a series of thirty-seven cases of acute putrid abscess of the lung treated surgically. All were operated on within the first six weeks of the disease, one half in the first three weeks, and one half in the second three weeks. Most of the operations were performed in one stage, adhesions being firm enough to wall off the pleural cavity. Adhesions are absent in abscesses situated near the interlobar fissure, and on the inferior surface of the lower lobe. The one-stage operative procedure was successfully employed in these cases also.

After exposure the abscess is "unroofed", drained, and packed. Complications are due usually to insufficient opening of the cavity or to adjacent extension of the suppuration. Such cases must be reopened.

Of the thirty-seven cases reported, thirty-six are living and all but one of these are symptom-free with negative x-ray and bronchoscopic findings.

In conclusion it may be said that putrid abscess is the only lung abscess of clinical importance, that it is not uncommon, and that its only satisfactory treatment is immediate surgery.

The paper was discussed by Dr. Elliott C. Cutler, Dr. Frederick T. Lord, and Dr. Edward D. Churchill. Dr. Cutler expressed a variance of opinion relative to the pathogenesis of lung abscess, stating that although aspirational pathogenesis occurs in some instances, by far the greater majority are probably embolic in origin. He doubts the ability of anaerobic organisms to penetrate the usually resistant bronchial mucosa and set up an anaerobic suppurative process. In a survey of the literature and analysis of 1500 cases only thirty per cent were definitely postoperative in occurrence. Although one half of this thirty per cent followed tonsillectomy, tonsillectomy constitutes at least one half of the surgery in the United States. Tonsillectomy cannot be condemned of undue etiological importance. He does not believe in early operative procedures because the process is still in its acute phase at that time, with inadequate walling off of the abscess; pleural adhesions are not yet adequate; and some cases may heal spontaneously. He favors delaying operative procedures until the process has become more chronic with walling off of the abscess and the pleural cavity. Dr. Frederick T. Lord agreed with Dr. Neuhof in considering the pathogenesis as aspirational in most cases. In his experience at least two thirds of the cases occur after operations in which aspiration of infected material is a distinct possibility. The initial symptom of cough observed in most cases of pulmonary abscess is not the initial sign of embolism, in which pain is usually the first symptom. Pleural involvement with formation of adhesions in the early stage of the disease has not occurred invariably in the cases observed by Dr. Lord. He condemned the use of exploratory aspiration of lung abscesses, stating that such a procedure is not necessary for diagnosis, and is apt to precipitate a fatal hemorrhage due to perforation of a large vessel. He does not advocate the use of bronchoscopy in every patient, and believes in the use of lipiodol only to differentiate bronchiectasis and abscess. The best treatment in his experience has been surgical. He advises conservative treatment of cases as long as they are progressing satisfactorily, however, since one third of the cases will heal spontaneously, and the average operative mortality is twelve per cent. Abscesses of two months or more duration usually require surgical intervention.

Dr. Edward D. Churchill agreed with Dr. Neuhof in all the fundamental concepts of the disease. He expressed a divergence of opinion as to the best time for operative procedures, preferring to wait until the fifth or sixth week for firm adhesions to be formed. He uses the two-stage operation, and believes that this procedure minimizes the possibility of complications, allows a more adequate "unroofing", and lessens the chance of chest wall infection.

HARVARD MEDICAL SOCIETY

The Harvard Medical Society met Tuesday evening, November 12, at the Peter Bent Brigham Hospital, Dr. Milton J. Rosenau presiding.

Two cases were presented, the medical case by Dr. Leo Sullivan: A twenty-two year old male Greek was first seen nine and a half months ago, at which time he gave a history of five or six months of recurrent joint pains, affecting ankle, wrist, elbow, and shoulder joints, with associated weakness, insomnia, malaise, and anorexia. He had spent two and a half months in bed prior to his entry to the hospital, and had lost thirty to forty pounds during this period. Physical examination at that time showed him to be markedly undernourished, and to be suffering from generalized muscle and bony tenderness. The spleen was palpable four fingers below the costal margin, and there was questionable enlargement of the liver. Laboratory studies were normal except for a hemoglobin of sixty per cent, and a white count of 18,000.

During his stay he was given intravenous administration of typhoid vaccine, with subsequent relief of his pain. It was noted that tender subcutaneous nodules appeared on both forearms. He was discharged home with instructions to remain in bed, which he did not do. He suffered exacerbations of his symptoms and an increase in number and painfulness of the subcutaneous nodules. He was admitted to the Boston City Hospital where one of the nodules was excised, and the diagnosis of periarteritis nodosa made on the basis of the histological picture of this tissue. Following this he had an irregular course of exacerbations and remissions of symptoms, the nodules swelling and becoming painful when he was physically active, and subsiding during bed rest.

On entry he was found to have gained twenty-five pounds since he was last seen, and to have improved markedly as to his general condition. Small freely movable lymph nodes were present in the cervical and axillary regions; the spleen and liver were both palpable four fingers below the costal margin. There was generalized bony and muscular tenderness, and over his left leg there were reddish areas one half to one and one half centimeters in diameter which showed thickening of the skin. No nodules were present on the legs, although they could be felt on both arms. Laboratory findings were negative except for a hemoglobin of sixty-eight per cent. Biopsy of one of the subcutaneous nodes confirmed the diagnosis of periarteritis nodosa. Occasionally patients with this disease show pulmonary changes simulating tuberculosis, but the chest plates in this case were negative. The temperature and white blood count continued normal. He was again receiving intravenous administration of typhoid vaccine with relief of pain. Evanescent nodules appeared over his arms, persisting one to four days, with gradual disappearance. No eosinophilia was observed.

Dr. Marshall N. Fulton in discussing the case commented on certain similarities between the signs and symptoms presented by this patient on his first admission and those described in Felty's syndrome (infectious arthritis with splenomegaly). The subcutaneous nodules were at first believed to be the nodules occasionally observed in rheumatoid arthritis, although the distribution was not characteristic of the latter disease. Twenty per cent of patients with periarteritis nodosa have subcutaneous nodules, biopsy of which allows the diagnosis to be made. In the absence of these peripheral nodules the manifestations of the disease are so bizarre and confusing that the diagnosis is rarely made during life except in cases subjected to exploratory laparotomy in which nodules may be found in the intra-abdominal arteries. Some cases have extensive liver involvement and jaundice; others may have involvement of the meningeal arteries and show signs suggestive of meningitis.

Dr. Robert Bates presented a case of "man bite fever" from the surgical service. A forty-nine year old Greek hotel keeper entered with a swollen painful right hand, which had increased in size and painfulness since he was bitten by an inebriate one week previously. For a period of approximately forty-eight hours after the infliction of the wound there was little untoward reaction except for moderate swelling and pain locally. After this period of mild symptoms there was an abrupt onset of acute illness, with a rise of temperature to 102.4°F., prostration, and enormous swelling of the hand to three times its normal size. The whole hand was a dark purple hue except for the fifth finger which was white and exuded extremely foul smelling pus. There was no lymphadenopathy. The blood showed a leucocytosis of 19,400, with ninety-one per cent polymorphonuclear leucocytes. Blood culture was negative. Smear of the lesion showed a myriad of various organisms, with many staphylococci, streptococci, and gram-positive rods. Culture from the lesion revealed growth of anaerobic hemolytic streptococci only. The fifth finger was amputated but the process extended to the fourth finger, which also had to be removed. Since the operation the hand had shown marked improvement, although the temperature was still fluctuating between 100 and 102°F.

Dr. Elliott C. Cutler compared human bite wounds with gangrene of the lung. They show characteristically a period of twenty-four or forty-eight hours with little sign of infection, and then gradual onset of gas formation, with the presence of innumerable organisms. The Welch bacilli causing such infections seem to be of lower virulence than those encountered in other cases of gas gangrene, and these lesions are as a rule more easily controlled.

Dr. Rosenau, in introducing Dr. Frederick F. Russell, lecturer in Preventive Medicine and Hygiene at the Harvard Medical School, told of Dr. Russell's pioneer work in the study and control of typhoid and dysentery, and of his introduction of vaccination against typhoid fever. Dr. Russell presented the address of the evening, his topic being: "Recent Studies in Yellow Fever—a Virus Disease." Extensive studies of Aztec literature, and of the records of the Portuguese navigators, have proved that yellow fever was originally an African disease, and was first brought to the western hemisphere by the importation of Negro slaves to Guadeloupe. From this island it was carried over the Caribbean and to the mainland by the buccaneers. The possibility that the Aedes aegypti mosquito might be the carrier of the disease was first suggested by Carlos Finlay, although he never proved the fact conclusively. Carter studied a yellow fever epidemic in Louisiana, and established the existence of the "extrinsic period of incubation", which was very suggestive of the existence of an insect vector. The army commission under the direction of Walter Reed proved that the disease was carried by the Aedes aegypti mosquito, and devised a method for control.

Carter and Shannon found that the Aedes aegypti breeds only in artificial collections of water, due to the clumsiness of the larval forms, which easily fall prey to other larvae in natural waters. This mosquito is a poor flier, and experiences difficulty in crossing even a wide city street. It passes more readily from back yard to back yard and across narrow streets and alleys.

Davis found that the length of the "extrinsic incubation period" varies inversely with the temperature at which the mosquito is kept. This period is apparently the length of time required for the virus to pass from the gut to the salivary glands of the insect. Davis also proved that the virus does not multiply within the mosquito, but diminishes gradually in concentration throughout the insect's life. The mosquito is not injured by the existence of the virus in its body, and its natural life span of about six weeks is not shortened by infection.

Recent laboratory studies have shown that no less than twelve other species of mosquito are able to carry yellow fever, and at least one epidemic in Brazil has been traced to the Aedes scapularis. There is also a possibility that infected ticks can carry the disease, if they are crushed on the skin.

The researches of Noguchi into the etiology of the disease are well known. His work unfortunately was performed on patients suffering from infectious jaundice, and he isolated the Leptospiria responsible for that disease, and not the organism causing yellow fever. Stokes, Bauer and Hudson succeeded in infecting Indian monkeys with inoculations of blood from active cases of yellow fever. They also succeeded in perpetuating the virus for long periods of time in mosquitoes. More recently Max Tyler has infected mice by injecting the virus intracerebrally.

The European hedgehog has been found to be susceptible to the disease, and related species in South America and Africa are being studied, to determine whether these animals, acting as hosts for the bacilli, may be responsible for the continuance of the disease in these regions.

The distribution of yellow fever, previously so widespread, has been narrowed in recent years, until it now embraces a relatively narrow band on either side of the equator in South America and Africa.

Immunity conferred by an infection is lasting throughout life. The persistence of positive protection tests, after a lapse of seventy-five years from the infection, suggests that some focus of infection may be existent in the body, although no such focus has ever been demonstrated. Protection tests have shown that many subclinical infections of yellow fever escape positive diagnosis. Laboratory tests are not of aid in making the diagnosis in the first ten days of the disease, and clinical signs must be relied upon during this period.

The control of the disease is best effected by mosquito control in cities and thickly populated districts, but in sparsely settled, tropical regions such measures are not practical. Attempts at vaccination with dead virus have been uniformly unsuccessful, and it has been impossible to attenuate the virus sufficiently by animal passage. Recently, however, attempts at attenuation by growing the virus in tissue cultures seem to have been attended by some success.

Serum virus mixtures have been successfully used as vaccines in both Africa and South America, and this kind of vaccination has completely stopped infections among yellow fever laboratory workers.

THE NEW ENGLAND HEART ASSOCIATION

The first meeting of the New England Heart Association for the current year was held in the Ether Dome of the Massachusetts General Hospital October 28, 1935.

The meeting was opened by Dr. Paul D. White who presented a pathological specimen of chronic constrictive pericarditis. The patient was thirty-one years old and showed the classical signs and symptoms of Pick's Disease one year after an acute pericarditis of unknown origin: enlarged liver, ascites, engorged neck veins and but slight enlargement of the cardiac dullness without murmurs. There was fluid in both pleural cavities. She was too ill for operation. At autopsy the heart showed a closely adherent, greatly thickened pericardium about one-half centimeter thick. Dr. White believed that the patient could have been cured if Dr. Churchill and he had arranged to receive the patient for operation a few months earlier.

Dr. Sylvester McGinn spoke briefly of a triangular pad of fat that often is normally found at the left pericardial diaphragmatic angle outside of the pericardial sac. Because of its position it may cause confusion in accurately measuring the left border of the heart in soft x-ray films and because of this possible error, it must be kept constantly in mind. Orthodiagraphy easily avoids this error as does also physical examination. Dr. White spoke of his having had actually to testify in court concerning a case in which the size of the heart had been overestimated by the inclusion in the transverse diameter of 2 cm. of fat at the left pericardial diaphragmatic angle.

Two unselected cases of congenital heart disease were then presented to the audience by Dr. White for diagnosis. The first was a woman of sixty-four who had had a transient hemiplegia and who presented occasional extrasystoles, an enlarged heart, and a continuous humming murmur with a systolic thrill at the second interspace just to the left of the sternum. Dr. Samuel Levine correctly diagnosed this as a case of patent ductus arteriosus. The second patient was a child of four and a half years who tired easily, was dyspneic, had palpitation, and coughed frequently. There were a systolic thrill and a loud systolic murmur most marked in the third left interspace. The enlargement found by x-ray was thought by the roentgenologist to be consistent with a rheumatic heart. Electrocardiogram showed an s-a* tachycardia with a slight right axis deviation. At autopsy an interventricular septal defect was found. Dr. Paul Emerson correctly diagnosed this case at the meeting. Dr. White presented these problems, drawn at random, to counteract somewhat the impression that the diagnosis of structural defects in congenital heart disease is usually difficult or impossible, especially in children. He did agree, however, that in infants under six or eight months of age the diagnosis is far more difficult than in older children and adults.

Dr. McGinn then briefly reviewed the experience at the Massachusetts General Hospital in the past ten years as to the accuracy of diagnosis of congenital heart lesions. It was found that thirty-nine per cent of all the congenital heart cases had been either correctly diagnosed or the correct diagnosis suspected, and this was increased to sixty-three per cent if the entirely symptomless and signless valvular defects and coronary peculiarities were excluded. It was pointed out that in cases with congenital abnormality of the coronary arteries there may be inverted T waves in the electrocardiogram, that in cases of auricular septal defects there are large right auricle, right ventricle, and pulmonary artery and a small aorta, and that hearts with bicuspid aortic valves often develop subacute bacterial endocarditis but that there is nothing by which to diagnose the congenital heart lesion. Dr. White adds that there are now eight congenital cardiovascular defects that are diagnosable in adults and in children above infancy, provided sufficient care is taken in assessing all the signs; these are as follows: (1) patency of the ductus arteriosus, (2) interventricular septal defect, (3) interauricular septal defect, (4) the tetralogy of Fallot, (5) uncomplicated pulmonary stenosis, (6) coarctation of the aorta, (7) a right aortic arch, and (8) congenital heart block. In infants the diagnosis of idiopathic cardiac hypertrophy is also usually possible.

Dr. H. B. Levine spoke on pulmonary infarction complicating severe chronic mitral valve disease and

*Sinu-auricular.

reported five recent cases. It was found in a series of fifty-two cases of mitral stenosis studied that 61 per cent of those with failure (twenty-six cases) had pulmonary infarction (there were only four cases without failure who developed this complication) in contrast to only 25 per cent of the twenty-four cases of hypertensive heart disease with failure who had infarction in the lungs. The cases which were presented demonstrated the fact that in mitral stenosis with failure the development of pulmonary infarction makes the patient refractory to treatment and the prognosis bad. This common complication is frequently missed and should be suspected if any of the common signs and symptoms of pulmonary infarction occur and especially if the patient is not doing well. Dr. Soma Weiss pointed out the hopeless prognosis in patients with congestive failure who develop jaundice, and said that in Germany where more careful dissection of the whole body at autopsy is carried out than in this country thromboses in the veins of the legs are frequently found in cases of mitral stenosis.

Dr. H. B. Sprague discussed "A New Method in the Treatment of Paroxysmal Tachycardia." If the attacks of tachycardia are frequent and prolonged or if the patient also has organic heart disease, it may become absolutely essential to limit their incidence and duration. A case was presented of a surgeon of fifty-four who has pure rheumatic aortic regurgitation and has had increasingly frequent attacks of paroxysmal tachycardia over the past eight years. In spite of all the usual methods his attacks continued to become more frequent and to last longer, and he began also to have anginal distress during them. On one occasion an attack lasted seventy-two hours. Mecholyn failed to help and upon Dr. Soma Weiss's advice syrup of ipecac was administered. This produced very severe and prolonged nausea, and the attack ceased. Since this time the patient has frequently used this method with remarkable success and he has greatly reduced the duration of attacks, saving his heart many thousands of wasted beats. He uses two drachms of the syrup of ipecac at the onset of an attack. Dr. Weiss discussed the method briefly. It has been used for some time at the Boston City Hospital. It is better than other emetics because it contains alkaloids that act centrally on the vagal center as well as having a local irritant action. The long-continued nausea and the accompanying vagal stimulation have never failed to stop an attack.

Dr. R. E. Glendy discussed the normal precordial lead (Lead 4) of the electrocardiogram. The P-wave may be upright, inverted, or diphasic. The QRS complex usually has a total amplitude twice that in other leads. The Q-wave is usually inverted and the QRS complex is slightly longer in duration than in the classical leads. The T-wave is inverted but within very wide limits of amplitude. Dr. A. Graybiel presented the abnormal lead 4. This lead may give the earliest evidence of coronary thrombosis when the ST segment is diphasic or displaced far downwards or the only evidence of old infarction at the apex of the left ventricle when the Q-wave disappears. In posterior wall infarctions, there is usually little or no change in lead 4. A shallow T-wave in lead 4 may be caused by digitalis, especially a high ST interval. Generally lead 4 abnormalities are in agreement with those in the classical leads. Occasionally lead 4 fails to show an abnormality when it is evident in the other leads, and vice versa. Dr. White in conclusion pointed out that the precordial electrode should be applied just median to the apex, that lead 4 is occasionally useful, but that it needs much further study.

WORCESTER DISTRICT MEDICAL SOCIETY

The regular monthly meeting of the Worcester District Medical Society was held at the Grafton State Hospital on Wednesday evening, November 13, 1935, with Dr. William F. Lynch in the chair and Dr. Erwin C. Miller as secretary.

Dr. Harlan Paine was host at an excellent buffet supper, which was served in the main auditorium prior to the meeting. After disposing of routine business, Dr. William Lynch turned the duties of the presiding officer over to Dr. Paine, the Superintendent of the Grafton State Hospital.

Dr. Paine then presented the President of the Massachusetts Medical Society, Dr. Charles E. Mongan, who was enthusiastically welcomed by all the members present. Dr. Mongan called the attention of the members of the Society to the medical aspects of the Social Security Act. He also reminded them of the very excellent work and interest in the State Society by former doctors in Worcester County and urged each member of the Society to take an active interest in medical and civic affairs.

Dr. William R. Morrison, Chairman of the Committee of Arrangements for the next Annual Meeting of the State Society, was introduced. He asked for the advice and support of the Society's members in making the next Annual Meeting a most successful one and requested that the dates of the Annual Meeting and dinner in Springfield next June 8, 9 and 10 be reserved for attendance at the meeting. Dr. Morrison said that a most excellent scientific and commercial program is being prepared for the Springfield meeting.

Dr. Benjamin Cohen, Research Member of the Grafton State Hospital Staff, spoke on "Repression Communicability in Catatonic Stupor." He was followed by Dr. Bardwell Flower, Assistant Superintendent of the Grafton State Hospital, who talked on "Parenteral Paraldehyde."

The meeting adjourned at 10 P.M. There were 130 doctors present at this meeting.

ERWIN C. MILLER, M.D., *Secretary.*

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The semi-annual meeting of the Essex North District Medical Society will be held at the Riverside Tavern, Haverhill, on Wednesday, January 8, 1936,

at 12:30 P.M. The entire program will be in charge of Dr. Charles E. Mongan, President of the State Society, and will be entirely devoted to medical economics.

E. S. BAGNALL, *Secretary.*

MASSACHUSETTS MEMORIAL HOSPITALS

There will be a meeting of the Surgical Section in the Ladies' Aid Room, Talbot Memorial, 82 East Concord Street, Boston, on Friday, December 20, 1935, at 12 noon.

Papers will be presented by Dr. Harry J. Lee and Dr. Kenneth Christophe.

MILO C. GREEN, *Secretary.*

GREATER BOSTON BIKUR CHOLIM HOSPITAL

Greater Boston Bikur Cholim Hospital medical meeting Monday evening, December 23, at 8:15 o'clock, at the Nurses' Home, 45 Townsend Street, Roxbury. Speaker: Dr. Soma Weiss. Subject: "Seven Aspects in the Management of Chronic Patients." The profession is invited.

HENRY BAKER, M.D., *Secretary.*

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held at the Peter Bent Brigham Hospital, Boston, Mass., Monday, January 6, 1936, at 8:15 P.M.

PROGRAM

I. Business: Consideration of revision of Constitution and By-Laws.

II. Clinical:

1. A Method of Determining Blood Volume. Dr. J. G. Gibson, 2nd.
2. Blood Volume Changes in Congestive Heart Failure. Dr. William Evans.
3. Concerning the Mechanism of Hypertensive Crises. Dr. Michel Pijoan.
4. Hemopericardium as a Cause of Sudden Death. Dr. Marshall N. Fulton.
5. The Early Diagnosis of Aortic Stenosis. Dr. Samuel A. Levine.
6. Aortic Stenosis, Angina Pectoris, and Syncope. Dr. A. W. Contratto.
7. Is Digitalis Present in Body Fluids in Digitalized Patients? Dr. Maurice A. Schnitker.

All members and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, DECEMBER 23, 1935

Tuesday, December 24—

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

Thursday, December 26—

*8:30-9:30 A.M. Clinic, Surgical Staff of the Peter Bent Brigham Hospital, at the Peter Bent Brigham Hospital.

Saturday, December 28—

*10-12. Staff rounds at the Peter Bent Brigham Hospital.

*Open to the medical profession.

December 20—Massachusetts Memorial Hospitals, Surgical Section. See notice elsewhere on this page.

December 23—Greater Boston Bikur Cholim Hospital. See notice elsewhere on this page.

January 6—New England Heart Association. See notice elsewhere on this page.

January 10—William Harvey Society. 8 P.M., Beth Israel Hospital, Boston.

February 24 to May 16, 1936—International Medical Postgraduate Courses in Berlin. See page 1211, issue of December 12.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

January 8—See page 1271.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

January 8—Wednesday. Danvers State Hospital, Hathorne. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Hoskins. Subject to be announced later.

February 5—Council Meeting, Boston.

February 12—Wednesday. Addison Gilbert Hospital, Gloucester. Clinic 5 P.M. Dinner 7 P.M. Speaker and subject to be announced later.

March 4—Wednesday. Lynn Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Timothy Leary. Subject: Arteriosclerosis.

April 1—Wednesday. Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Richard H. Overholt of the Lahey Clinic. Subject: Chest Surgery.

May 7—Thursday. Censors' Meeting.

May 13—Wednesday. Annual Meeting. Salem Country Club. Dinner at 7 P.M. Speaker: Dr. Paul White. Subject to be announced later.

R. E. STONE, M.D., Secretary.
88 Lothrop Boulevard, Beverly.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings are held on the second Tuesday of January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Meetings to be held at the Bear Hill Golf Club at 12:15 P.M.

January 8.
March 11.
May 6.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

January 28, 1936—Hotel Kenmore at 8 P.M. Subject: "Compulsory Sickness Insurance." Speakers to be announced.

February 25, 1936—Massachusetts Memorial Hospitals at 8 P.M. Papers by the staff.

March 31, 1936—Hotel Kenmore, at 8 P.M. Dr. Benedict F. Boland—"Cauterization of the Cervix Uteri Using Various Electrical Methods." Illustrated with lantern slides.

May, 1936—Annual Meeting. (Place, date and subject to be announced.)

The censors meet for the examination of candidates: May 7, 1936; November 5, 1936.

FRANK S. CRUICKSHANK, M.D., Secretary.
1236 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January 16—Goddard Hospital. Subject and speakers to be announced later.

March 19—Plymouth County Sanatorium, South Hanson.

April 16—Brockton Hospital.

May 21—Lakeville State Sanatorium.

G. A. MOORE, M.D., Secretary.
167 Newbury Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 29, 1936—Joint Meeting with the Boston Medical Library at 8 Fenway. "Observations Around the World," Dr. Walter B. Cannon.

March 18, 1936—Meeting at the Boston Medical Library. "The Laboratory and Clinical Story of Fatigue," Dr. Arlie V. Bock and Dr. David B. Dill. Discussion: Dr. Donald J. MacPherson and Dr. Augustus Thorndike, Jr.

April 29, 1936—Annual Meeting at the Boston Medical Library. "The Treatment of Septicaemia," Dr. Champ Lyons. "The Pleurality of Scarlatinal Streptococcus Toxin," Dr. Sanford B. Hooker. Discussion: Dr. Hans Zinsser.

The medical profession is cordially invited to attend all of these meetings.

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary,
FRANCIS T. HUNTER, M.D.,
Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

January 8, 1936—Wednesday evening. Worcester City Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

February 12, 1936—Wednesday evening. Worcester State Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

March 11, 1936—Wednesday evening. Memorial Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

April 8, 1936—Wednesday evening. Hahnemann Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

May 13, 1936—Wednesday afternoon and evening. Annual Meeting of Society. Time, place and details of program to be announced in an April issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

Puerperal Gynecology. J. L. Bubis. 199 pp. Baltimore: William Wood & Company. $3.50.

A Treatise on Medical Jurisprudence. Benton S. Oppenheimer. 290 pp. Baltimore: William Wood & Company. $4.00.

Awaken Your Sleeping Beauty. Lilyan Malmstead. 96 pp. New York: E. P. Dutton & Company, Inc. $1.75.

Public Health Administration in the United States. Wilson G. Smillie. 458 pp. New York: The Macmillan Company. $3.50.

Diseases of the Thyroid Gland. Arthur E. Hertzler. Third Edition, Entirely Rewritten. 348 pp. St. Louis: The C. V. Mosby Company. $7.50.

The Evaluation of Symptoms. Offered after fifty years in medicine. Oliver T. Osborne. 163 pp. New Haven: The Yale University Press. $3.50.

A Text-Book of Fractures and Dislocations. Covering their pathology, diagnosis and treatment. Kellogg Speed. Third Edition, Thoroughly Revised. 1000 pp. Philadelphia: Lea & Febiger. $11.00.

Food and Beverage Analyses. Milton Arlanden Bridges. 246 pp. Philadelphia: Lea & Febiger. $3.50.

Free Medical Care (Socialized Medicine). 360 pp. New York City: Noble & Noble, Publishers, Inc. $2.00.

Laboratory Methods of the United States Army. Fourth Edition. Edited by James Stevens Simmons and Cleon J. Gentzkow. 1091 pp. Philadelphia: Lea & Febiger. $6.50.

The Treatment of Diabetes Mellitus. Elliott P. Joslin. 620 pp. Philadelphia: Lea & Febiger. $6.00.

Body Water. The Exchange of Fluids in Man. John P. Peters. 405 pp. Springfield: Charles C Thomas. $4.00.

The Sexual Relations of Mankind. Paolo Mantegazza. 335 pp. New York: Eugenics Publishing Company. $6.00.

Diseases of the Nose and Throat for Practitioners and Students. C. J. Imperatori and H. J. Burman. 723 pp. Philadelphia: J. B. Lippincott Company.

The Pathology of Internal Diseases. Second Edition. William Boyd. 904 pp. Philadelphia: Lea & Febiger. $10.00.

Modern Home Medical Adviser. Your Health and How to Preserve It. Edited by Morris Fishbein. 905 pp. New York: Doubleday, Doran & Company, Inc.

Mechanics of Normal and Pathological Locomotion in Man. Arthur Steindler. 424 pp. Springfield and Baltimore: Charles C Thomas. $8.00.

Pediatric Treatment. A manual of the treatment of the diseases of infants and children designed as a reference work especially for the General Practitioner and Physicians entering the field of pediatrics. Philip S. Potter. 578 pp. New York: The Macmillan Company. $5.00.

Individual Exercises. Selected exercises for individual conditions. George T. Stafford, Harry B. DeCook, and Joseph L. Picard. 111 pp. New York: A. S. Barnes & Company. $1.00.

Diabetes, The Malady of Our Time, A Manual. Etiology, courses and treatment of "Diabetes Mellitus". Aspects of diabetes mellitus in the light of modern research. Dr. Levy-Lenz and Dr. Heinz Schmeidler. 64 pp. Berlin: A. K. Verlag. $1.15.

Radium Treatment of Skin Diseases, New Growths, Diseases of the Eyes, and Tonsils. Francis H. Williams. 118 pp. Boston: The Stratford Company. $2.00.

The Stomach and Duodenum. George B. Eusterman, Donald C. Balfour, and others. 958 pp. Philadelphia and London: W. B. Saunders Company. $10.00.

A Textbook of Bacteriology. Thurman B. Rice. 551 pp. Philadelphia and London: W. B. Saunders Company. $5.00.

BOOK REVIEWS

Bernardo de Gálvez in Louisiana. 1776-1783. John Walton Caughey. 290 pp. California: University of California Press.

In this valuable monograph the author gives in "Part One, The Background," a complete account of the establishment of Spanish control in Louisiana and takes the reader in part two through Gálvez's administration and the Spanish struggle for domination of the Mississippi and the capture of Mobile.

After the capture of Pensacola, the author tells us that Gálvez was ordered to undertake the conquest of Jamaica, but after many delays and contretemps, we are told that "Rodney's victory over DeGrasse brought the whole project to a standstill."

Gálvez returned to Spain, coming back to the New World in 1784 as Captain General of Cuba, but soon he was sent to Mexico City to succeed his father as Viceroy of New Spain. His rule was short, however,

as he died on November 30, 1786, apparently of yellow fever. He was then thirty-eight years old. He was mourned by all. "New Spain would praise him, and sing of a perfect Viceroy, a most upright Judge."

There is much of interest in this monograph to American physicians, who will enjoy the well-documented and accurate account of the Spanish occupation of Louisiana.

1000 Questions and Answers on T. B. Edited by Fred H. Heise. 232 pp. New York City: National Tuberculosis Association.

This book of some 200 pages consists of a group of questions and answers relating to every phase of the tuberculosis problem, grouped in sixteen chapters.

It will prove of distinct value not only to the public at large but to the medical profession desirous of getting accurate information and advice from a well-recognized authority on the subject concerning any point relating to tuberculosis.

The index makes it possible to find the answer easily and quickly.

The need for books of this type which are short, compact, low-priced and yet authoritative is a very real one. Such a book as this is welcome.

International Clinics. Volume II. Forty-Fifth Series, 1935. Edited by Louis Hamman. 327 pp. Philadelphia, Montreal and London: J. B. Lippincott Company.

As in preceding volumes, this one is marked by a group of excellent articles by authors well known for their contributions in their respective fields and well worth reading. Dr. Hamman in addition to editing this fine volume has also contributed a very interesting article.

The Medical Man and the Witch during the Renaissance. Gregory Zilboorg. Third Series, Volume II. 215 pp. Baltimore: The Johns Hopkins Press. $2.50.

This volume consists of three lectures given before the Institute of the History of Medicine, Johns Hopkins University. It is the second of the Hideyo Noguchi lectures, the first of which, "The Renaissance of Medicine in Italy", by Arturo Castiglioni, was published in 1934 and reviewed in The *New England Journal of Medicine*, March 15 of the same year.

Dr. Zilboorg, a psychiatrist, presents a topic of unusual interest. For many years he has been studying the history of psychiatry and these lectures testify to his scholarship and spirit of investigation. The first lecture concerns itself largely with an account of a book, the "Malleus Maleficarum", first issued in the last years of the fifteenth century. This volume became the leading and practically the only textbook on the Inquisition and, before 1669, was printed ten times. It thus was a popular and useful book for its time, for it "proved" the existence of witchcraft and witches and had behind it the authority of the church of Rome. Upon the basis of this book men assumed the power of judgment and many hundreds of people were burned as witches in the sixteenth century. A second lecture covers more definitely the relationship between witchcraft and medicine, and the third lecture is given over entirely to an account of Johann Weyer, the founder of modern psychiatry. Zilboorg's account of Weyer is by far the finest in existence and much of the value of this work for future historians lies in his excellent account of this man.

The book is heavy with quotations and facts, the style is not light, and the volume cannot be recommended for popular reading. For the serious student of medical history, however, it will have a definite appeal and it forms a worthy addition to the publications already issued in this series.

Historie Des Universités Françaises et Etrangèrs. Stephen D'Irsay. Tome II. Du XVIe Siècle a 1860. 457 pp. Paris: Auguste Picard.

The first volume of this interesting history of the universities was reviewed in The *New England Journal of Medicine*, February 1, 1934. Before the second volume could be issued the author died a tragic death in Paris, November 16, 1934. Estimates of his importance to the history of medicine will be found in the *Bulletin of the Institute of the History of Medicine*, Johns Hopkins University, for June 1935. The second volume of his work, which is now at hand, has, therefore, been issued posthumously. The manuscript, however, was finished before the author's death and it was only the setting up and printing which had to be supervised. This has been splendidly done and the volume is in every way comparable with the first part of the work issued a year ago. In addition, one finds as an appendix a list of the manuscripts consulted, an extensive biographical index of inestimable value to scholars, and a complete index to both volumes.

The Evaluation of Symptoms. Offered after fifty years in medicine. Oliver T. Osborne. 163 pp. New Haven: The Yale University Press. $3.50.

Solidly packed with precious material, important and useful, this little volume, privately printed by the author, should be on every doctor's desk and in every intern's coat pocket. With his wide experience Osborne is certainly the philosopher to discuss a considerable group of symptoms and their value to the clinician. As he well remarks only the consultant can ignore the complaints of the patient. Many a medico has built his reputation, and no mean one, on an uncanny ability to understand and relieve the complaints of his charges. And both care little whether a diagnosis is ever made. In this little book, which should be followed by others of its kind, there is nothing stale or irrelevant. Old truths are emphasized and new ones clarified. Rarely has a work given the reviewer so much genuine pleasure and profit.